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PATHOGENESIS OF INTERMITTENT EXOPHTHALMOS

FRANK B. WALSH, M.D., AND WALTER E. DANDY, M.D.

BALTIMORE

Intermittent exophthalmos is a rare, but striking and unmistakable, syndrome. It is characterized by pronounced and rapid—almost instantaneous—protrusion of one eye when venous stasis is induced by bending the head forward; by lowering the head; by turning the head forcibly; by hyperextension of the neck; by coughing; by forced expiration, with or without compression of the nostrils, and by pressure on the jugular veins. The ocular protrusion disappears immediately when the head is erect and when artificially induced venous congestion is relieved.

Usually, but not invariably, there is enophthalmos when venous congestion does not obtain. There may or apparently may not (to judge from cases reported in the literature) be pulsation of the eyeball. Vision may or may not be affected. The condition is progressive and may be productive of unbearable pain and troublesome diplopia. The appearance is unsightly, but life is not at stake.

The case here reported—the only one we have seen—is presented because the pathologic features were disclosed at operation. Most previous reports of cases are mainly clinical presentations, and the clinical study is not followed by detection of the underlying lesion. Orbital operations have been performed in a few cases, and venous beds have been described in the orbit. These may or may not represent the whole pathologic picture. No postmortem examinations have been made.

REPORT OF A CASE

A white girl aged 18 was referred by Dr. Milton Little, of Hartford, Conn., with the complaint of bulging of the left eye induced by posture and associated with pain in the head.

The family and past histories were without significance.

Present Illness.—From the age of 10 years the patient had known that her left eye was sometimes more prominent and at other times less prominent than her right eye. When she sat erect or stood, the left eye became sunken. When she lay down, it bulged. When she became excited, it protruded. For six months, every morning on awakening, there had been a throbbing pain in the eye, which disappeared soon after she got up. The pain disappeared after rest in bed for a week, but on resumption of her usual activities it soon returned. For a week there had been severe pain in the left eye and the left temple. The throbs were noted to occur at the same rate as the heart beat. From the age of 10 years there had been occasional periods when diplopia was present, but strabismus or ptosis had not been observed.

Examination.—When the patient stood, there was striking enophthalmos. Immediately after she lay down, the eye commenced to protrude, and pronounced exophthalmos persisted throughout the recumbent period. Moreover, the eyeball pulsated; this was visible and palpable. When she leaned forward (head down) the exophthalmos became extreme. This was also true when the jugular veins were compressed or when she blew her nose. The exophthalmos was also increased when the common carotid artery was compressed (because the internal jugular vein was then also compressed), but the pulsation ceased. There was no audible bruit.

There were weakness of the left external and inferior rectus muscles and absence of ptosis when the patient was sitting erect. When the eye was protruded as a result of change of position of the head, ptosis appeared. Measurements were made with the Hertel exophthalmometer. There was enophthalmos of 3 mm. (fig. 1 *A*) when the head was erect (in the sitting or standing position the measurement for the right eye was 15 mm. and for the left eye 12 mm.). There was exophthalmos of the left eye: 6 mm. when she was lying recumbent (fig. 1 *B*); 11 mm. when the head was tilted forward or the jugular vein was compressed (fig. 1 *C*); 18 mm. when expiration was forced and the nostrils were compressed (patient standing; fig. 1 *D*); 5 mm. when the head was turned forcibly to the left, and 3 mm. when it was turned to the right. Visual acuity was 20/15 in the right eye and 20/30 in the left eye (with the patient sitting upright). The left pupil dilated slightly when the eyeball protruded. The eyegrounds were normal except for slight overfilling of the retinal veins of the left eye when that eye was maximally protruded.

Roentgenograms showed thickening of the superior margin of the left orbit, which was increased in density, as was the left wing of the sphenoid. The sphenoid fissure was widened (fig. 2). A small, diffuse area of calcification was visible in the outer orbit (fig. 2).

Diagnosis.—1. The quick protrusion and sinking of the eyeball with the postural changes, and the rapid protrusion induced by coughing, sneezing and jugular compression could only mean filling of a large venous bed.

From the Johns Hopkins Hospital and University.

2. The pulsation of the eyeball indicated an arterial component. The lesion, therefore, had to be an arteriovenous aneurysm.

3. The absence of a murmur indicated that the communication between arteries and veins was through vascular "coils," and not through a fistula.

4. The enophthalmos (with the patient sitting or standing) was thought to be due to atrophy of the orbital fat from long-continued pressure.

which the brain was free of attachment to the dura. The cerebral vessels were unaffected and of normal size. In the region of the sphenoid fissure and the anterior part of the middle fossa was a mass of coiled vessels (fig. 3); this overflowed the sphenoid wing and extended a short distance over the orbital plate (about 1 cm.) as a thin, pink film (of arterial blood), which blanched with slight pressure. The mass was intimately grown into, and was inseparable from, the dura and was

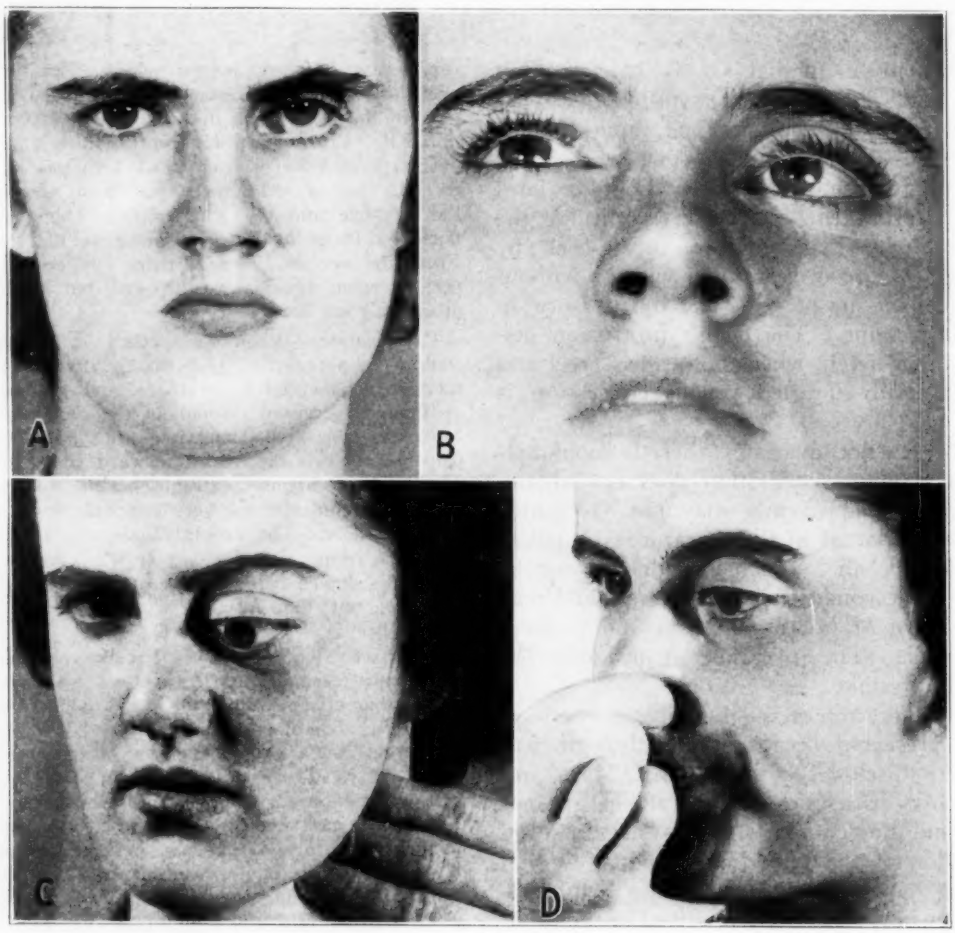

Fig. 1.—*A*, patient in the erect position, showing difference in the palpebral fissures. There is pronounced enophthalmos on the left side. *B*, degree of exophthalmos when the patient was recumbent. *C*, exophthalmos and downward protrusion of the eyeball from pressure on the jugular vein. *D*, exophthalmos induced by forced expiration when the nose was closed.

5. The most puzzling point was the absence of exophthalmos when the patient was erect. Coiled vessels could be expected to produce a space-occupying mass in the orbit. The explanation of this was obtained at operation. The coils making a space-occupying mass were in the cranial chamber, and not in the orbit.

Operation (W.E.D.).—On July 24, 1943, a frontotemporal approach to the cranial chamber was made—a typical hypophysial approach. When the dura was turned back, a finger-like projection of vascular coils was observed extending from the region of the pterion backward and mesially, and finally paralleling the sylvian vein. A few minor vascular attachments to the brain were thrombosed with the electrocautery, after made up of intertwining coils of vessels, easily compressible, like a sponge, and immediately returning to the original size when pressure was released. The vascular mass was repeatedly attacked by the electrocautery, and the volume gradually shrank until the scar was flush with the sphenoid fissure.

From time to time the cautery cut through the vessels, and brisk arterial bleeding occurred. The bleeding areas were covered with pieces of muscle, and the coagulation was carried out through them. The sphenoid fissure was greatly widened and filled with the vascular coils, but no attempt was made to carry the cauterization forward into the orbit, there being no reason to believe that a mass was located therein. Be-

cause of diffuse dural bleeding, the middle meningeal artery was isolated and coagulated at the foramen spinosum. It was our impression that the arterial component of the arteriovenous aneurysm arose from this vessel because of its intimate relation to the coiled mass and because no other large artery was nearby. The intracranial division of the internal carotid artery was distant and was entirely normal. The optic nerve was in full view, was not implicated and was not injured at any time. The aneurysmal mass completely covered the dura over the gasserian ganglion and the cavernous sinus. It could not be determined whether or not there were communications with the latter. That there were no communications with the internal carotid artery in the cavernous sinus cannot be stated dogmatically, but this could be possible only through an anomalous branch. It was realized throughout the operation that the nerves to the extraocular muscles lying in the cavernous sinus and the sphenoid fissure were passing through the vascular mass that was being coagulated, and that their injury, or even destruction, was a distinct possibility. However, the cure of the aneurysm was thought to be advisable in view of the progressively increasing pain.

Postoperative Course.—Immediately after operation the eyeball was free of all the intermittent changes that previously obtained with posture, blowing the nose, jugular compression, etc. However, vision was entirely lost in the left eye, and all the extraocular muscles were paralyzed. The pupil did not respond directly or consensually to stimulation with light. Three months later the intermittent exophthalmos remained cured, but the extraocular paralyses persisted in part. The pupil exhibited a consensual reaction. There was enophthalmos of 3 mm. The upper lid could be elevated incompletely, and internal and downward rotation of the eyeball had improved to about half-normal. The price of a cure was therefore high, but the patient is happy over the end result. We have wondered whether it would have been practical and preferable to have attempted ligation of the orbital veins in the orbit (through the same transcranial approach) and to have left the aneurysm untouched. The practicability of this procedure is uncertain, but even if the operation had been successful, the pulsation of the eyeball would have persisted.

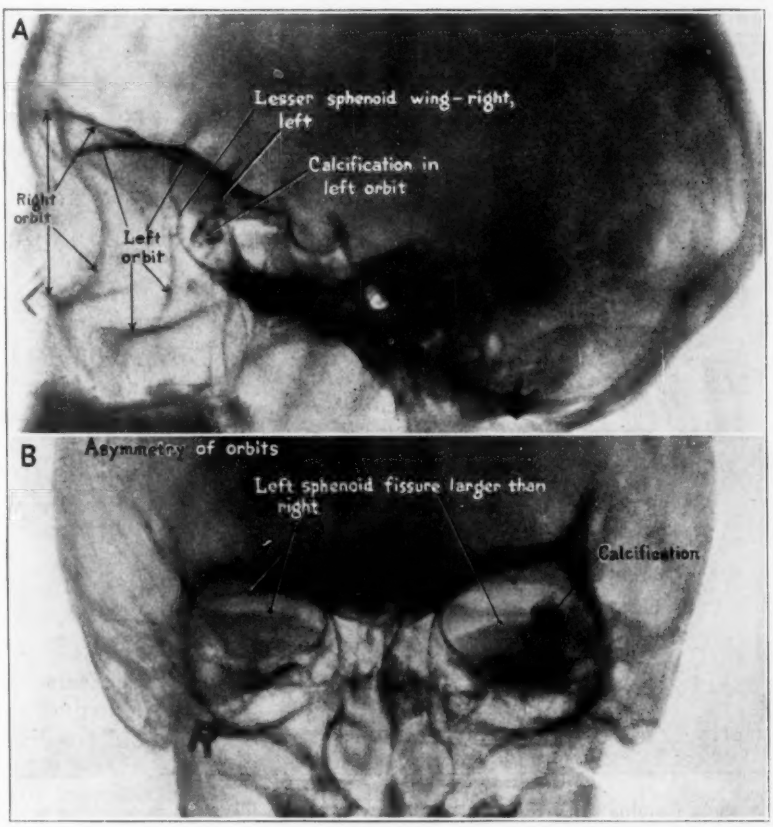

Fig. 2.—Retouched roentgenograms of the orbit, showing area of calcification and widening of the left sphenoid fissure.

NOTE.—The patient was seen again ten months after discharge from the hospital. There had been no discomfort except on one occasion, three weeks before reexamination, when she had a severe pain in the head. She stated that at this time there was some protrusion of the left eye.

Examination revealed incomplete ptosis and inward deviation of the eye of about 30 degrees. She was unable to elevate the left eye but could lower it 20 to 30 degrees. The eye was blind and the nerve atrophic. There was enophthalmos of the left eye, which was not influenced by such factors as position and pressure on the jugular vein.

SYMPTOMATOLOGY AND BRIEF REVIEW OF LITERATURE

Intermittent exophthalmos has been recognized as a clinical entity since 1805, when it was first described in an infant. In a classic paper, Birch-Hirschfeld reviewed the literature up to 1906 and presented observations of prime importance concerning not only the intermittent exophthalmos but the anteroposterior position of the normal eye in relation to various positions of the head. He described the syndrome in a physician, Dr. Minor, who also wrote regarding it. Wissmann and Schulz (1922) added a single case and collected the cases appearing between 1906 and 1922, a total of 74. Kraupa and Mendl (1936) described a case and brought the number reported in the literature to 96 cases. They rejected 2 cases included by Wissmann and Schulz.

The following reports of cases of intermittent exophthalmos have been found since Kraupa and Mendl's publication, and the list also includes 2 cases not mentioned by them: Bartók (1931), Chapman (1931), de-Petri (1935), Marchesini (1935), Hippert (1936), Muirhead (1936), Lipovich (1936), Petrov (1939), Spektor (1939), Giqueaux (1942), Poole (1942), Dunphy (1942) and Rones (1942). The cases reported by the two last-named authors were mentioned in the discussion on Poole's paper. Rychener (1942) and Ellett (1940) made subsequent reports on Chapman's case. These, with our case, bring the total number of cases to 111.

From the clinical viewpoint little has been added since Birch-Hirschfeld's comprehensive review.

Occurrence.—As the foregoing figures indicate, true intermittent exophthalmos is rare. Birch-Hirschfeld saw a single case among 150,000 patients with ophthalmic disease, and de Schweinitz, 1 case among 100,000 such patients; the case described here is the only one of the condition recorded at the Johns Hopkins Hospital. Krauss, Hippert and Weisner each encountered 2 cases. In the United States and Canada, the condition has been described by Sattler, de Schweinitz, Byers, Chapman, Ellett, Poole, Dunphy, Rychener and Rones.

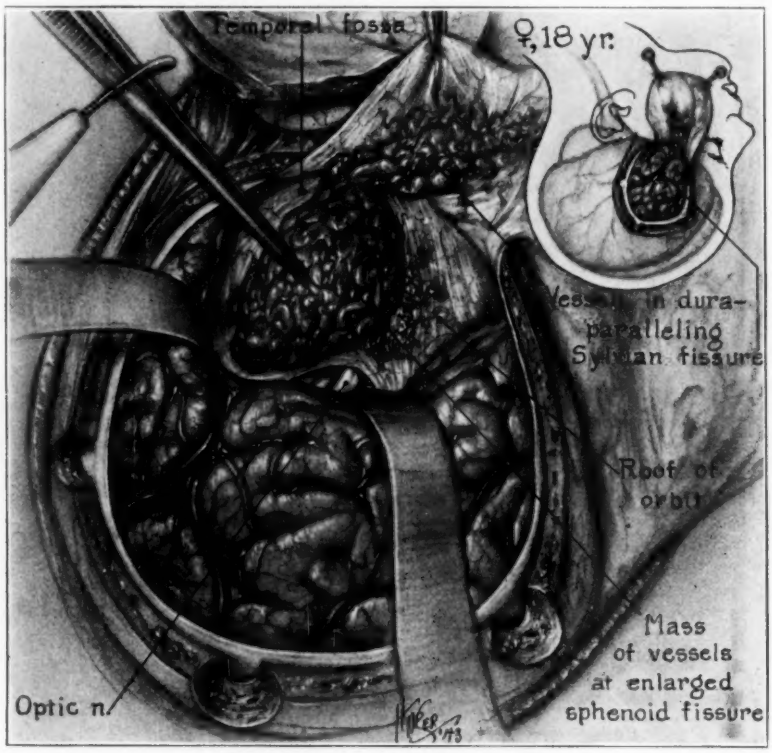

Fig. 3.—Coil of vessels forming an arteriovenous aneurysm which filled the floor of the middle fossa and overflowed around the sphenoid wing into the dura of the anterior fossa. These vessels contained arterial blood and pulsated.

Sex.—Possibly more men than women are victims of this condition. Birch-Hirschfeld's series contained 35 men and 15 women. In

Kraupa and Mendl's series 10 patients were males and 8 females.

Trauma.—Trauma has been suggested as a factor in the development of intermittent exophthalmos, but it is difficult to believe it can play more than a precipitating role. The patient described by Rumjanzewa was first observed to exhibit the syndrome during childbirth.

Age of Onset.—Since persons with intermittent exophthalmos may be wholly unaware of its existence until it has been pointed out to them, it is impossible to obtain accurate information regarding the age of onset. The condition has been seen in infants, and it has been observed for the first time during the sixth decade of life (Birch-Hirschfeld). Sattler expressed the opinion that it occurred in young persons almost, or quite, exclusively, but there is ample evidence that this concept is erroneous. Birch-Hirschfeld found records of 6 cases occurring in the first decade of life, 7 in the second, 8 in the third, 1 in the fourth, 2 in the fifth and 1 in the sixth. Of cases reported since 1935, 3 have occurred in the second decade of life, 4 in the third, 5 in the fourth and 2 in the fifth.

Unilaterality.—In all reported cases the condition has been unilateral. Poole commented that the left eye is involved ten times as often as the right. This high ratio is not upheld by Birch-Hirschfeld's compilations, although he stated that the left eye was affected more often than the right. Kraupa and Mendl found the right eye affected in 10 cases and the left eye in 8 cases. In the 13 cases described since 1935, the left eye was always affected. Excellent accounts of intermittent exophthalmos of the right eye have been given by Byers and Rumjanzewa.

It is natural that attempts have been made to explain the predominance of involvement of the left eye. Reese suggested that the jugular foramen on the left side is frequently smaller than that on the right, but this structure is remote from the lesion producing the venous changes and can have no bearing on its causation. There is no apparent reason that a congenital lesion, such as that causing this condition, should favor one side.

The Exophthalmos: Its Direction and Degree.—The complete syndrome of alternating exophthalmos and enophthalmos usually develops gradually and progressively. Radswitzki described a case of exophthalmos which occurred only as a result of severe coughing or vomiting up to the age of 14 years, but thereafter a change in position of the head, holding the breath, etc., was sufficient to induce the condition, which rapidly disappeared when the head resumed the erect position. Mulder described the case of a man who until the age of 12 years exhibited intermittent exophthalmos only when he wore a tight collar but later presented the complete syndrome. Mention has already been made of Rumjanzewa's case, in which the anomaly developed during the strains of childbirth.

The eye may be protruded either directly forward or downward and outward. In a majority of reported cases mention is made only of direct protrusion. According to Birch-Hirschfeld, the superior ophthalmic vein is usually larger than the inferior ophthalmic and, consequently, the eye is usually pushed downward and outward, as well as forward. The position of the eyeball is doubtless dependent on the symmetry or asymmetry of the venous bed in the orbit.

Narrowing of the palpebral fissure during the episodes of exophthalmos was observed in Poole's and Rumjanzewa's cases and in our case. During the stage of enophthalmos the palpebral fissures on the two sides were quite, or almost, equal in width. Probably Dunphy's suggestion that during exophthalmos there may be voluntary or reflex closure of the lids as a protective measure is correct.

The amount of proptosis has been noted to vary within wide limits in different cases. Sattler and de Vincentiis observed 25 and 29 mm. respectively in their cases when the head was bent forward. In our case, 18 mm. of relative exophthalmos was observed when the patient exhaled forcibly with the nostrils compressed, but this did not represent what could have been obtained, since it was thought unwise to attain the maximum. It may be remarked that measurements of exophthalmos as usually made are only approximate; special photographic apparatus, such as that described by Birch-Hirschfeld, is necessary for accuracy.

The time required for proptosis to begin is not stated in most reported cases. Birch-Hirschfeld observed a latent period of five seconds, followed by protrusion of the eye, which reached almost its maximum in thirty seconds and its full maximum in another fifteen seconds. His patients had the head bent forward. He commented that the latent period had been stated as being from one to five minutes but that it rarely, if ever, is as long.

Factors Influencing Exophthalmos.—Often, as in our case, the condition has first been observed by a friend. Birch-Hirschfeld observed protrusion of the normal eye when the head was bent forward. This, of course, is due to gravity and is intensified when an abnormal venous bed exists.

Proptosis can be produced in most instances by pressure over the jugular vein on the side of the intermittent exophthalmos. In our case this produced about as much proptosis as did bending the head forward. Occasionally, as in Lindenmeyer's case, pressure over the contralateral vein produced proptosis when similar pressure over the homolateral jugular vein failed to do so. In such instances, either there is great congenital narrowing or absence of the homolateral jugular vein, or it has become thrombosed.

The position of the head in lateral rotation has been found to influence the position of the eye, owing to a degree of jugular constriction. Mann observed that rotation of the head to the right produced maximal venous drainage through the left jugular vein, and, conversely, rotation of the head to the left produced maximal venous drainage through the right jugular vein. In our case forcible turning of the head produced exophthalmos of the affected eye, which was more pronounced when the head was turned to the left. In Poole's photographs the effect is most striking.

Birch-Hirschfeld surmised that any obstruction to the anterior venous pathways outside the orbit resulted in additional pressure in the ophthalmic veins, and, to prove it, he devised an ingenious experiment by which the extraorbital veins were compressed. This, however, could have no bearing on the intermittency of the exophthalmos. Krauss assumed that large congenital varicosities in the orbit might cause intermittent exophthalmos. He also assumed that an obstruction to the drainage of orbital blood, either anteriorly, as suggested by Birch-Hirschfeld, or posteriorly, could produce intermittent exophthalmos. Both these observers commented on the narrowness of the ophthalmic vein just before it enters the cavernous sinus, and Krauss remarked on there being a pronouced constriction of the superior ophthalmic vein where it lies close to the tendon of the superior oblique muscle. However, an obstruction to the veins is the one thing that cannot explain this condition; the prompt appearance of proptosis with jugular compression means that there is no obstruction in the venous channels. All textbooks are in agreement that there are no valves along the ophthalmic vein. There could not, of course, be any effective valves in intermittent exophthalmos because pressure on the jugular veins immediately produces the ocular protrusion.

Enophthalmos.—Enophthalmos is not essential to the diagnosis, but it is usually present. It can only be due to absorption of orbital fat from pressure of the vascular bed (probably venous) in the orbit. Enophthalmos, when present, is apparent only when the head is in the erect position.

Lids, Temporal Region and Face.—It might well be expected that there would be some engorgement, or at least prominence, of the veins of the eyelids, temple and face on the side of the anomaly, since they are in communication with the orbital veins. However, this is only occasionally observed and was not present in our case. Some swelling of the lids may be observed when the eye is proptosed. The temporal region was engorged during proptosis in Marchesini's case, and the homolateral side of the face was swollen in Petrov's case. In Rumjanzewa's case there was flushing of the side of the face when the eye was protruded.

In no instance, so far as we are aware, has there been great dilatation of veins over the face and lids or over the scalp, as is so commonly observed in cases of carotid-cavernous fistula and pronounced cirsoid aneurysm.

Facial Asymmetry.—In several instances retardation of growth of the homolateral side of the face has been described, but Birch-Hirschfeld concluded that this was not of particular significance because of the relative frequency of asymmetry of the face in otherwise normal persons. There can be no local reason for asymmetry of the face.

The Affected Eye.—It is obvious from consideration of reported cases that the involved eye in a great majority of instances remains normal. When the eye is proptosed and remains so for a considerable time, there may be congestion of the bulbar conjunctiva. In a relatively small number of the reported cases some degree of optic nerve atrophy (14 per cent, Birch-Hirschfeld) has been present. In our case vision was slightly reduced —20/30. Birch-Hirschfeld assumed that optic nerve atrophy was due to retrobulbar hemorrhage, which is said to be, and doubtless is, an occasional complication. However, a more reasonable assumption in a majority of such cases would appear to be long-sustained direct pressure of the mass on the optic nerve.

Visual acuity may be lowered materially during the exophthalmic phase. Extreme overfilling of the retinal veins has been noted during the period of exophthalmos, and they may pulsate; but the fundus remains otherwise unchanged. Diplopia, present occasionally in our case, is not usually mentioned. Usually there is no limitation of ocular movements. The pupil remains unchanged in many cases, but pupillary dilation during exophthalmos has been described by several observers listed by Birch-Hirschfeld and was present in our case. Rumjanzewa described

narrowing of the pupil during periods of exophthalmos in her case.

Pulsation of the Eyeball.—According to Birch-Hirschfeld, this symptom is rarely present in cases of intermittent exophthalmos. Mention of it was made in only 7 of 74 cases included in his study and in that of Wissmann and Schulz. The symptom was present in our case and was most pronounced during maximum exophthalmos. It is entirely possible that the pulsation was overlooked in some of the reported cases in which it was not mentioned. However, in many cases special mention has been made of the absence of pulsation, and there can hardly be a doubt that it is not always present. In only 1 instance has a subjective murmur or objective bruit been noted. In Delord and Viallefont's case a blowing sound was heard on auscultation. Poole's patient described roaring and tinnitus. Neither was present in our case despite the known arteriovenous communication.

Other Symptoms.—In many cases the condition is asymptomatic; but pain may be the only symptom, and it may become steadily more severe. Since pain is associated with protrusion of the eye, it may be present most of the time. A sensation of fulness in the side of the face is frequently noted. In Poole's case the pain became so severe that the patient could not continue his work. Often there is complaint of dizziness and vertigo, usually not severe and occurring only during periods of exophthalmos. Birch-Hirschfeld, in a short discussion (1930), suggested that intracranial varices possibly account for these symptoms. Hippert expressed the opinion that venous anomalies influenced the vestibular apparatus in his case.

Roentgenograms.—Roentgenographic evidence has usually been reported as negative. Hippert noted a single small calcification in the sphenoid fissure, but there was a similar one on the other side. Kraupa and Mendl reported small calcifications, which they termed phleboliths, in 2 cases. Gastreich saw seven or eight rounded and slightly oval, smooth, pea-sized shadows. Lyding reported twelve such shadows in the orbit in his case. Such shadows are well known to occur in the defective walls of cerebral vascular beds, such as arteriovenous aneurysms in the cranial and orbital cavities, and doubtless these calcifications are of similar origin. In our case there were two calcified plaques in the outer orbit (fig. 2).

Treatment and Pathologic Features.—Rychener (1942) observed improvement in a case reported by Chapman (1931) and later by Ellett (1940).

The injection of sclerosing solutions into the orbital veins has been advocated by several authors: Hippert noted successful results in 4 cases; Ravardino reported a success, but Dunphy, a failure.

The first operative attack on the large veins of the orbit for intermittent exophthalmos was made by Schimanowsky (1907). Through an incision under the eyebrow, he clamped and twisted the large vein, perhaps the superior ophthalmic, in the back of the orbit and allowed the clamp to remain two days before being withdrawn. Cure of the exophthalmos resulted, but with ptosis and ophthalmoplegia; the state of vision was not given. Three years later he operated in another case in similar fashion except that he tied and cut the veins. Precisely the same result was obtained. Löwenstein (1911), through a modified Krönlein approach, palpated venous tortuosities on the way to the supra-orbital vein. Bleeding was severe; it was controlled by the Paquelin cautery, and the wound was packed. During the next few days the exophthalmos was extreme but gradually disappeared by the twelfth day; four days later there was an enophthalmos of 3.5 mm., and this persisted. Slight exophthalmos (1.5 mm.) persisted during jugular compression. The inferior rectus muscle was paralyzed; vision was greatly reduced immediately after operation but returned to 4/10 in four weeks and to 10/10 in nine weeks.

Germain and Weill explored the orbit of a patient who had had a retrobulbar hemorrhage and exhibited pronounced exophthalmos. A large number of dilated veins were observed within the muscle cone. In the attempt to isolate and ligate these veins there was spontaneous hemorrhage. Excision of the veins was performed. The exophthalmos was relieved. Two months later, the eye was enophthalmic, and there was external ophthalmoplegia. The state of vision was not recorded.

The only other case in which operation was done was reported by Rumjanzewa (1930), and a perfect result was obtained. The ophthalmic vein was ligated in the orbit through a supra-orbital incision; the exophthalmos was cured, without extraocular palsies. The author called it Golowin's operation—a method of attack used by him on pulsating exophthalmos.

In none of these records of operations is there any mention of pathologic observations except that the veins were large. The exposure of the orbit is doubtless too restricted to permit definition of any clearcut lesion; on the other hand, there is probably nothing in the orbit except large veins. The operation used in our case, as previously detailed, was a transcranial approach

which has been evolved for orbital tumors. Since the lesion was intracranial and of a well recognized type, and since it was obliterated intracranially, there was no indication for removal of the orbital roof to inspect the orbit. This approach revealed the only extraorbital lesion that has been identified with intermittent exophthalmos. Had the orbit been explored by a Krönlein approach, the character of the underlying lesion would not have been disclosed. This arteriovenous aneurysm (not a single fistula, but coils of vessels replacing capillaries, with a larger lumen and of congenital origin) is of exactly the same character as are all arteriovenous aneurysms within the substance of the brain. Several cases of these aneurysms are cited in a paper published by one of us in 1928; two photographs of such aneurysms are included here (figs. 4 and 5).

We do not suggest that the treatment carried out in our case is the best solution of the problem. On the contrary, the result obtained by Rumjanzewa is so much better that in another case we should probably be content to ligate the superior ophthalmic vein and leave the aneurysmal coils alone. The surgical attack is much better, and there is consequently less risk of injuring the extraocular muscles through the transcranial than through either the Krönlein or the frontal approach. The ultimate decision concerning the best type of surgical treatment will, of course, be evolved only after greater experience and after it is known whether lesions of different types may be responsible for this unusual syndrome.

However, there is a serious risk in ligating large veins ahead of an arteriovenous aneurysm. In 1 of our cases of cerebral aneurysm (fig. 4) another nearby vein ruptured. It must be realized that the great venous bed emerging from the arteriovenous coils is made as an adjustment to the entering arterial blood, and reduction in the bed throws a strain on the remaining veins, all of which have relatively weak walls.

COMMENT

In our case there could be no preoperative doubt that an arteriovenous aneurysm was causing this syndrome, i. e., in view of the pulsation of the eyeball. Nor could it be doubted that it was a "coiled" rather than a carotid-cavernous aneurysm (a fistula), which gives entirely different signs and symptoms, i. e., a murmur, constant exophthalmos and dilated, pulsating vessels in the conjunctiva and over the forehead. There are never alternating exophthalmos and enophthalmos with a fistula, nor does jugular compression affect the protrusion of the eye. The coexisting enophthalmos was new in our experience but was assumed to be due to atrophy from pressure of the dilated veins. It is noteworthy that the space-occupying mass, i. e., the vascular coils, were almost entirely in the cranial chamber, and not in the orbit. If this mass, as large as a walnut, had been in the orbit, there would have been constant exophthalmos, and

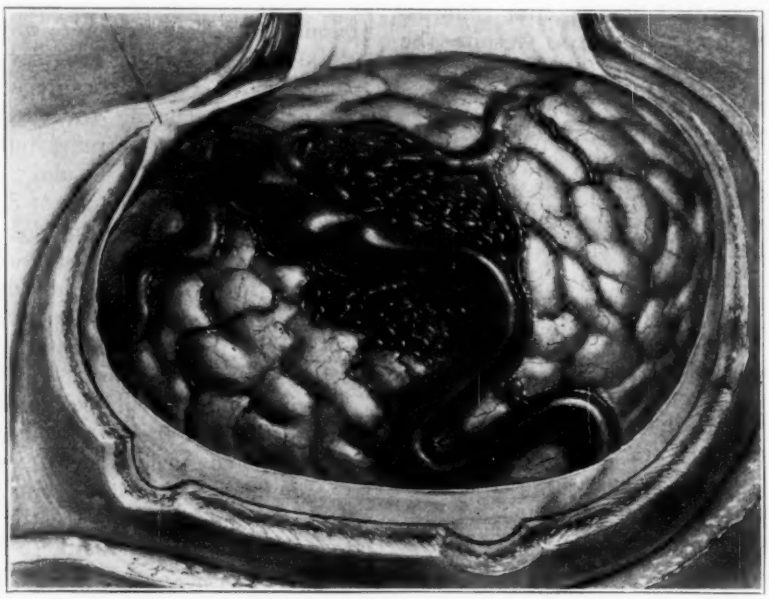

Fig. 4.—Arteriovenous aneurysm of the brain, fundamentally similar to that presented in this paper (from Dandy, 1928, fig. 4).

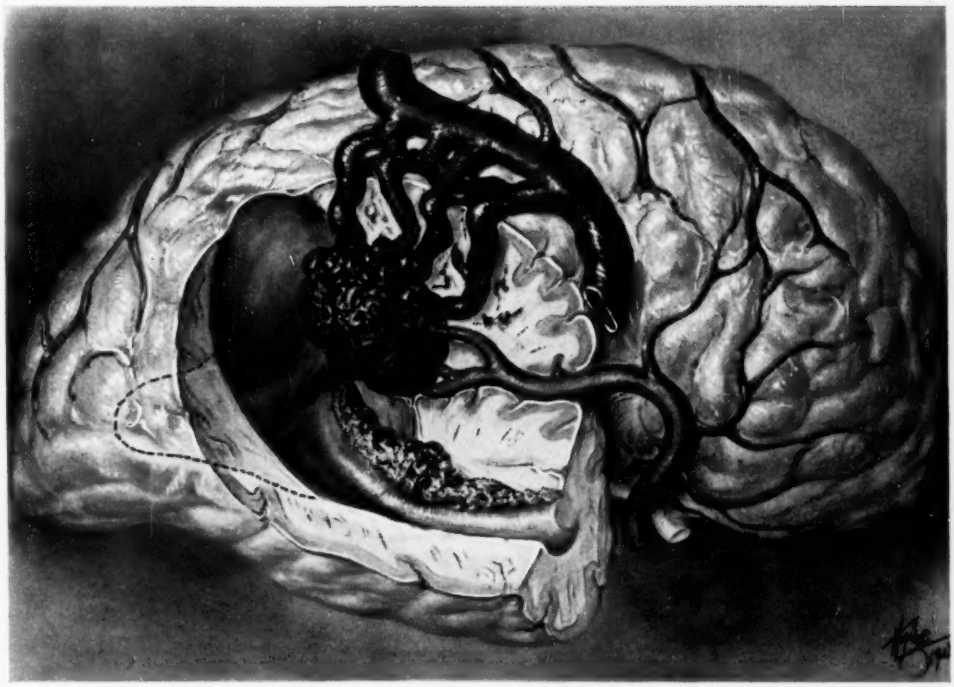

Fig. 5.—Reconstruction of an arteriovenous aneurysm, showing a coil of vessels which carry arterial blood and from which the tremendously enlarged veins emerge (from Dandy, 1928, fig. 14). This drawing is shown because it is exactly like the arteriovenous aneurysm described in this paper.

never enophthalmos. Only the enlarged veins that drained the aneurysm were in the orbit, and the size of these vessels changed with the volume induced by gravity and other physiologic factors affecting venous pressure largely through the corresponding jugular vein.

A glance at figures 4 and 5 will show the tremendous effect of arterial pressure on the size of the veins emerging from a cerebral arteriovenous aneurysm; the effect on the vein from an orbital or extraorbital aneurysm of similar type will be of precisely the same character.

It is not clear how far the pathologic changes in this case can be applied in all cases of intermittent exophthalmos. In all cases with pulsation of the eyeball there is doubtless just such a causative lesion. But in many, perhaps in the majority, of reported cases the pulsation either was not present (as determined by special mention of its absence) or was missed by the examiner. At least there must always be a large venous bed, but we know of no description of such large venous masses in the orbit. While purely venous aneurysms occur in the brain, they are uncommon, and they are not known to enlarge progressively. A true angioma would not be quickly influenced by changes in jugular pressure but would act simply as a space-occupying mass, producing a constant exophthalmos.

Until there are more pathologic confirmations of the underlying lesions in this condition, a conclusion applicable to all cases is clearly impossible.

It is possible that there are two general types of lesions causing intermittent exophthalmos, (1) arteriovenous with pulsation and (2) venous without pulsation; but speculation on this score is useless. The existence of the arteriovenous type at least is certain; that of the venous type is problematic. It is our guess that ocular pulsation has been overlooked in many cases. It is never so pronounced as in the usual pulsating exophthalmos from a carotid-cavernous fistula.

Arteriovenous aneurysm is, of course, a congenital maldevelopment, and never of traumatic origin. This accounts for its early appearance in most recorded cases. It might be asked why it is not present at birth. The answer is that the sustained arterial blood pressure causes the gradually progressive venous enlargement as the vascular coils with defective walls steadily dilate. The exact time of appearance of the syndrome, therefore, depends on the resistance of these walls, which must have wide individual variations. Doubtless many open in later life.

CONCLUSIONS

In a typical case of intermittent exophthalmos the underlying lesion was observed at operation—a transcranial approach—to be an intracranial arteriovenous aneurysm lying in and behind the sphenoid fissure. This case is the only one in the literature in which a cause for this rare syndrome has been disclosed. The source of the arterial part of the aneurysm is not certain; it was thought to be the middle meningeal artery.

An arteriovenous aneurysm of similar type is probably responsible in all cases for pulsation of the eyeball. In most recorded cases pulsation was absent or missed. Whether or not there are two types of this syndrome, one with and the other without pulsation, cannot be determined without subsequent pathologic studies. Doubtless, in some cases the pulsation was present but was missed by the observer.

The intermittent exophthalmos was cured by obliterating the aneurysm with the electrocautery, but blindness of the affected eye and ophthalmoplegia resulted.

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KERATECTOMIES FOR TREATMENT OF CORNEAL OPACITIES

RAMÓN CASTROVIEJO, M.D.
NEW YORK

The surgical treatment of corneal opacities has been in use for over two centuries. Boury,[1] in 1743; Plenck,[2] in 1783; Bertrandi,[3] in 1798; Wenzel,[4] in 1808; Demours,[5] in 1821; Rosas,[6] in 1830; Carron du Villards,[7] in 1838; Chelius,[8] in 1839; Cappelletti,[9] in 1844, and Desmarres,[10] in 1847, spoke of scarifications and abrasions of the cornea. Small, nonperforating punctures made with a knife or needles in the cornea in order to provoke inflammatory processes tending to clear up the opacities were advocated by Ware,[11] in 1795; by Holchers,[12] in 1842, and by Himley,[13] in 1843. Riveri,[14] in 1836, advocated paracentesis of the anterior chamber to improve the vision of eyes with corneal opacities, especially those with abundant vascularization.

Another surgical procedure used to improve vision in eyes affected with corneal opacities consists in the excision of the superficial lamella of the opaque cornea. This method was advocated by Chevalier Taylor[15] in the early part of the eighteenth century; by Boury,[1] in 1743; by Platner,[16] in 1747; by Pellier,[17] in 1783; by Gouan,[18] in 1783; by Bell,[19] in 1788; by Malgaigne,[20] in 1843; by Cappelletti,[9] in 1846; by Guepin,[21] in 1847, and by Denovillers[22] and Gosselin,[23] in 1855. Others who, without advocating this method, described it are: Duddell,[24] in 1729; Mead,[25] in 1775; Scarpa,[26] in 1809, and Mackenzie,[27] in 1854. In 1846 Donders[28] studied the regeneration of corneal tissue after excision, reporting that when one half or two thirds of the superficial layers of the cornea are removed, healing occurs without inflammation or any other abnormality.

Some of the methods of corneal excision described by the aforementioned authors resemble remarkably surgical procedures which have been described in the past thirty or forty years. Those who have been interested in the excision of cor-

From the Institute of Ophthalmology, Columbia-Presbyterian Medical Center.

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3. Bertrandi, cited by Ovio, G.: L'oculistica di Antonio Scarpa, Naples, V. Idelson, 1936, vol. 1, p. 294.

4. Wenzel, cited by Ovio, G.: L'oculistica di Antonio Scarpa, Naples, V. Idelson, 1936, vol. 1, p. 294.

5. Demours, cited by Ovio, G.: L'oculistica di Antonio Scarpa, Naples, V. Idelson, 1936, vol. 1, p. 294.

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7. Carron du Villards, cited by Ovio, G.: L'oculistica di Antonio Scarpa, Naples, V. Idelson, 1936, vol. 1, p. 295.

8. Chelius, cited by Ovio, G.: L'oculistica di Antonio Scarpa, Naples, V. Idelson, 1936, vol. 1, p. 295.

9. Cappelletti, cited by Ovio, G.: L'oculistica di Antonio Scarpa, Naples, V. Idelson, 1936, vol. 1, p. 295.

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12. Holchers, cited by Ovio, G.: L'oculistica di Antonio Scarpa, Naples, V. Idelson, 1936, vol. 1, p. 295.

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28. Donders, F. C.: Untersuchungen über die Regeneration der Hornhaut, Holländ. Beitr. zu d. anat. u. physiol. Wissensch. **1**:387-400, 1846-1848.

neal scars in more recent years are: Jobson,[29] in 1909 and 1912; Wiener,[30] in 1909, 1926 and 1939; Zentmayer,[31] in 1909; Rucker,[32] in 1929; Goar,[33] in 1931; Hilgartner Sr. and Jr.,[34] in 1931 and 1937; Castroviejo,[35] in 1937; Verhoeff,[36] in 1939, and James,[37] in 1942.

Still another surgical procedure advocated for the treatment of corneal opacities is corneal transplantation. Since the number of surgeons who have written about the surgical treatment of corneal opacities is large, it will not be possible to describe in this paper their operative technics. Readers who are interested in more detailed information about the previous work on this subject are referred to the original publications of the authors mentioned.

Among the corneal opacities covering the pupillary area there are some susceptible of treatment by corneal transplantation, which gives the best results as far as improvement of vision is concerned. Other opacities are unfavorable for keratoplasty on account of excessive density of the scar or pronounced superficial vascularization, which would render the transplant nebulous or opaque. Corneas affected by dystrophia adiposa and other forms of dystrophy are unfavorable for keratoplasty because the disease process invades the transplant, rendering it nebulous or opaque.

There are other superficial opacities in the pupillary area which, although lending themselves to satisfactory treatment by corneal transplantation, are best handled by other surgical procedures which expose the eye to fewer complications. Recurrent pterygium, pseudopterygium and symblepharon, especially when they have invaded the pupillary area, do not lend themselves well to corneal transplantation.

For some of these conditions for which corneal transplantation is not indicated, superficial keratectomy is the preferred procedure. Keratectomy may be partial, when only a limited area of the external lamella of the cornea is excised, or total, when the excision extends over the whole area of the cornea.

It is not the purpose of this article to give statistics for patients subjected to keratectomies; this will be the object of a future publication. At present I shall limit myself to a brief description of the different types of keratectomy which I have been using for the past few years, justifying this preliminary report by the usefulness which this type of surgical treatment may have in repairing the ocular sequela of war injuries.

The illustrations used in this article have been taken from moving pictures of the operations. The photographs illustrating the different steps of the operations have been retouched in order to eliminate confusing reflexes and shadows. The photographs illustrating the postoperative results are not retouched.

1. PARTIAL SUPERFICIAL KERATECTOMY FOR THE TREATMENT OF BAND KERATITIS

This operation may be carried out with either general or local anesthesia. After the speculum is inserted, the eye is fixed with O'Brien or Elschnig forceps. The area of the cornea to be excised, including the opacity extending across the pupillary area, is outlined by two incisions made with the Graefe knife above and below the opacity, the incisions penetrating approximately one-third the depth of the cornea (*1*). The incisions outlined with the knife can be made more visible by the instillation of a 2 per cent solution of fluorescein sodium. Dissection of the superficial layers of the cornea, including the opacity, is started on the temporal side with the aid of the Graefe knife (*2*), the edge of the corneal flap being held with fine conjunctival forceps or, better yet, with jeweler's forceps, which are strong and have very fine points. The dissection of approximately the outer third of the corneal thickness, including the opacity, is carried out with the aid of a Graefe knife from the temporal to the nasal side, until the limbus is reached (*3*). At the limbus, the corneal flap thus dissected is excised with scissors (*4*), so that the pupillary area is left transparent (*5*). The operation is concluded with

29. Jobson, G. B.: Report of a Case of Nodular Opacity of the Cornea Cured by Excision, Tr. Am. Acad. Ophth. **14**:300-302, 1909; Keratectomy for Removal of Corneal Scars and Opacities, Ophth. Rec. **21**: 332-336, 1912.

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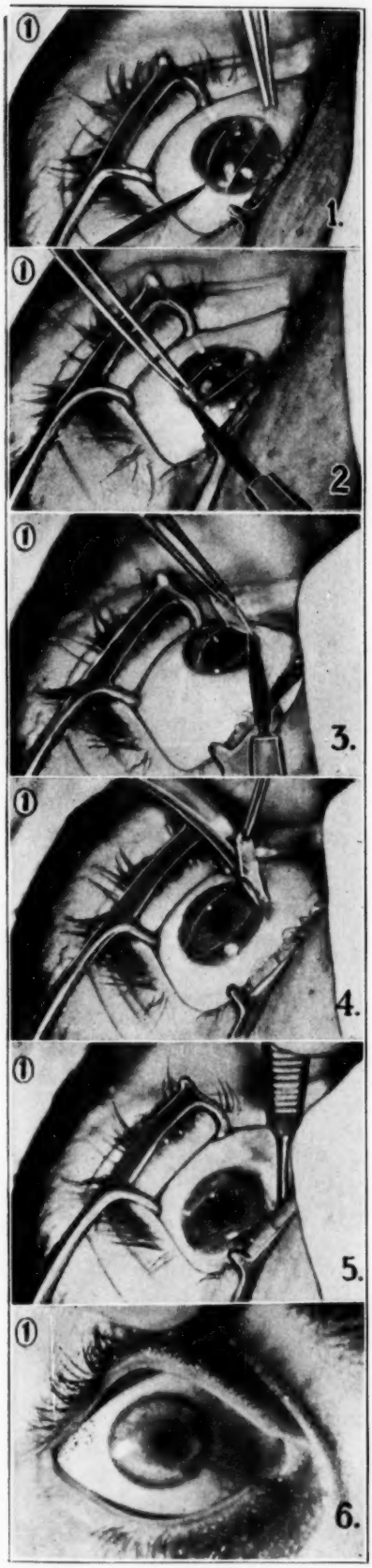


Fig. 1.—Steps 1 to 6.

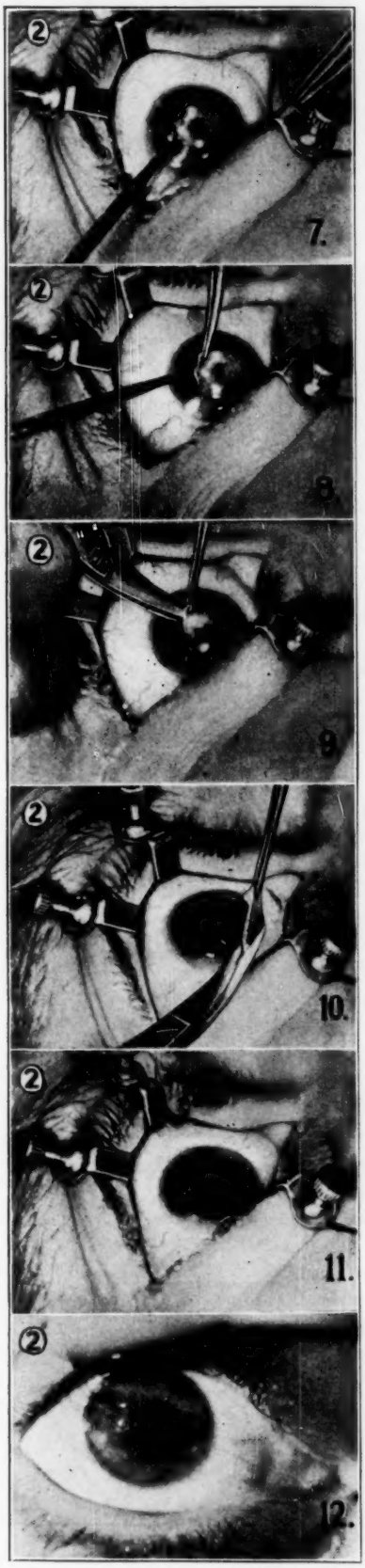


Fig. 2.—Steps 7 to 12.

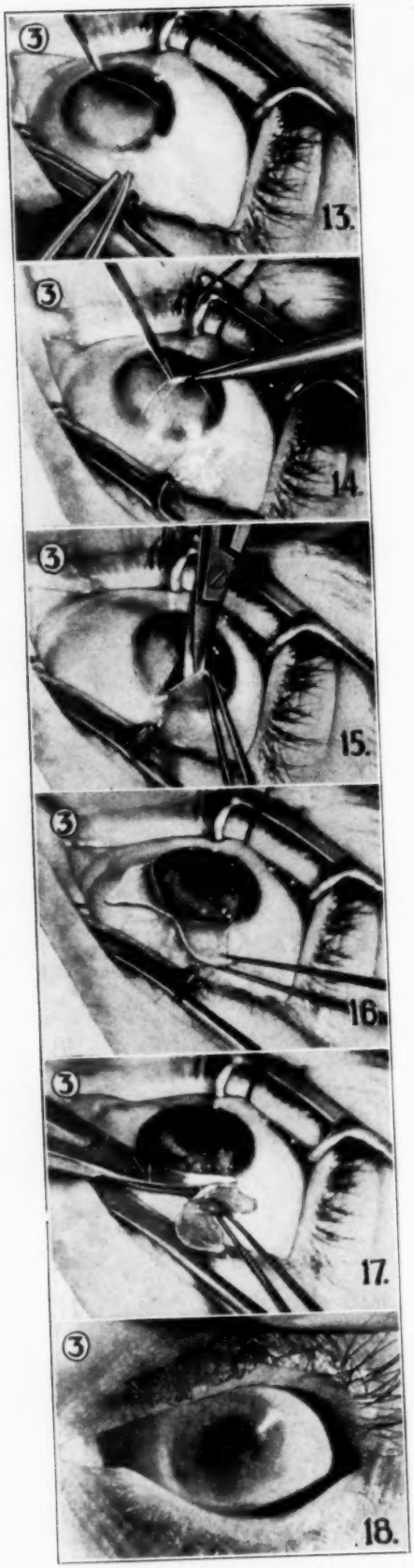


Fig. 3.—Steps 13 to 18.

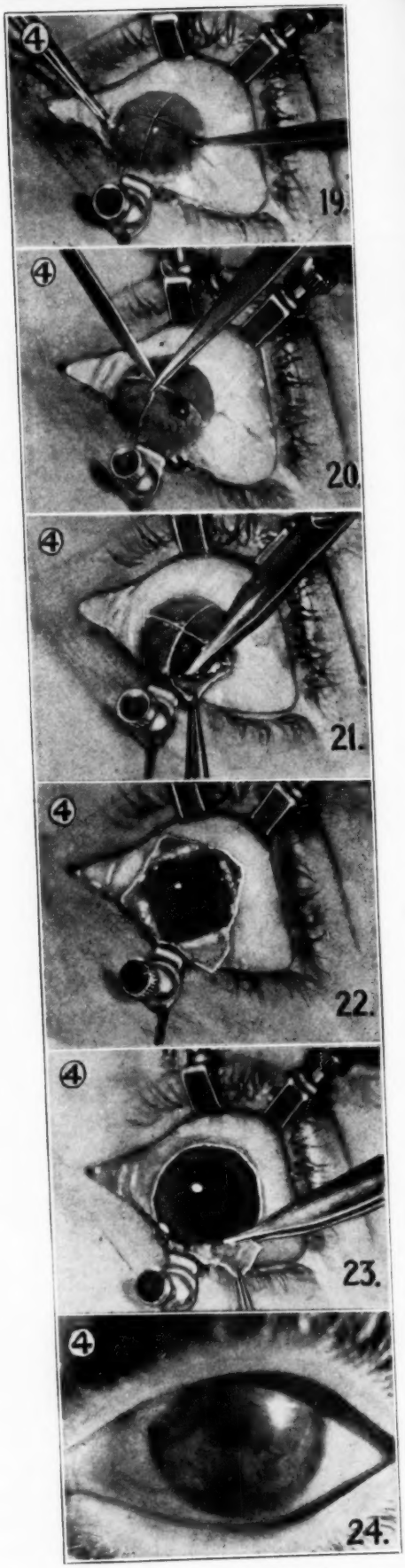


Fig. 4.—Steps 19 to 24.

the application of an antiseptic ointment, such as metaphen (1:3,000) or, better yet, penicillin ointment,[38] containing 500 Oxford units per gram, and a monocular dressing. The eye is dressed daily, with instillation of a 2 per cent solution of fluorescein sodium to observe the progress of the epithelization of the cornea, and the application of an ointment of metaphen or penicillin. If there is ciliary injection, it is advisable to instil atropine sulfate, in 1 per cent solution.

As a rule, the cornea is completely epithelized in from four to seven days, and the eye may be uncovered. In about two weeks the eye is quiet; the pupillary area remains transparent (*6*), and improvement of vision is considerable (from ability to see hand movements or count fingers at a few centimeters to an acuity of 20/200 or better). For several months after the operation there is likely to be considerable irregular astigmatism, which later tends to diminish.

2. PARTIAL SUPERFICIAL KERATECTOMY FOR TREATMENT OF DYSTROPHIA ADIPOSA CORNEAE

This operation may be performed with general or local anesthesia. After insertion of the speculum or of a substitute, such as the mosquito lid clamp retractors shown in the illustrations, the eye is fixed with forceps. The upper limit of the corneal area to be excised is outlined with the aid of the Graefe knife (*7*), the incision penetrating approximately one-third the depth of the cornea. The incision made with the knife is better visualized by the instillation of a 2 per cent solution of fluorescein sodium. The dissection of the superficial layers of the cornea, including the opacity, is started with the aid of the Graefe knife while the lip of the corneal flap is held with fine conjunctival forceps or with jeweler's forceps (*8*). With the aid of scissors with blunt points,[39] the dissection is extended to the limbus (*9*). The flap of cornea thus obtained, including approximately the outer third of the corneal thickness and the opacity, is excised at the limbus with the aid of scissors (*10*). The pupillary area thus uncovered contains only healthy, transparent corneal tissue (*11*). The operation concludes with the application of an antiseptic or a penicillin ointment and a monocular dressing.

The postoperative treatment is carried out as described in the previous operation. The results obtained with this method are similar to those obtained with the operation previously described for the treatment of band keratitis. Several weeks after the operation the pupillary area may be expected to be clear (*12*), with considerable improvement of vision. In these cases of dystrophia adiposa corneae it is advisable to carry out a thorough investigation of the patient's general condition to find out whether there is any systemic cause which might influence the recurrence of the ocular lesion. If any such cause is found, it should be corrected. The operation just described is also indicated for other types of corneal dystrophy involving only the external layers of the cornea.

3. PARTIAL SUPERFICIAL KERATECTOMY FOR TREATMENT IN LEUKOMA

Eyes favorable for this type of operation are those in which, as a sequel of infection or trauma, scars have developed over a limited area of the superficial cornea. The operation is similar to the two previously described and can also be undertaken with local or general anesthesia. After insertion of the speculum and fixation of the eye with forceps, a Graefe knife is used to limit the corneal area to be dissected (*13*). With the same knife, another incision is made perpendicular to the first one, in order to divide the area to be excised into two flaps (*14*). If the edge of the corneal flaps is held with the jeweler's forceps, from one third to one half of the external layers of the cornea (depending on the depth of involvement of the cornea by the leukoma) is dissected away, the incision being begun with a Graefe knife (*14*) and continued with scissors (*15*) until the limbus is reached. When the two flaps thus outlined have been dissected down to the limbus (*16*), they are excised at the limbus with scissors (*17*). The operation ends with the application of an antiseptic or a penicillin ointment. The postoperative course resembles that outlined for the operations previously described.

The improvement of vision obtained by this operation depends on the extent of the leukoma into the corneal stroma. A few weeks after the operation the eye is usually quiet, and the appearance (*18*) and vision are considerably improved. If, after excision of the leukoma by the technic of the partial keratectomy just described, vision should not improve as much as desired, the eye, which before the keratectomy was unfavorable for keratoplasty, is rendered more favorable for this type of surgical procedure.

4. TOTAL SUPERFICIAL KERATECTOMY FOR TREATMENT OF VASCULARIZED LEUKOMA

This operation may be performed on eyes in which superficial vascularized scars have developed after severe injuries or infections. The operation may be performed under local or general

38. The penicillin ointment, with a jelly base, was supplied by E. R. Squibb & Sons.

39. The special scissors used for this operation, and for subsequent operations, are manufactured by V. Mueller & Co., Ogden Ave., Chicago.

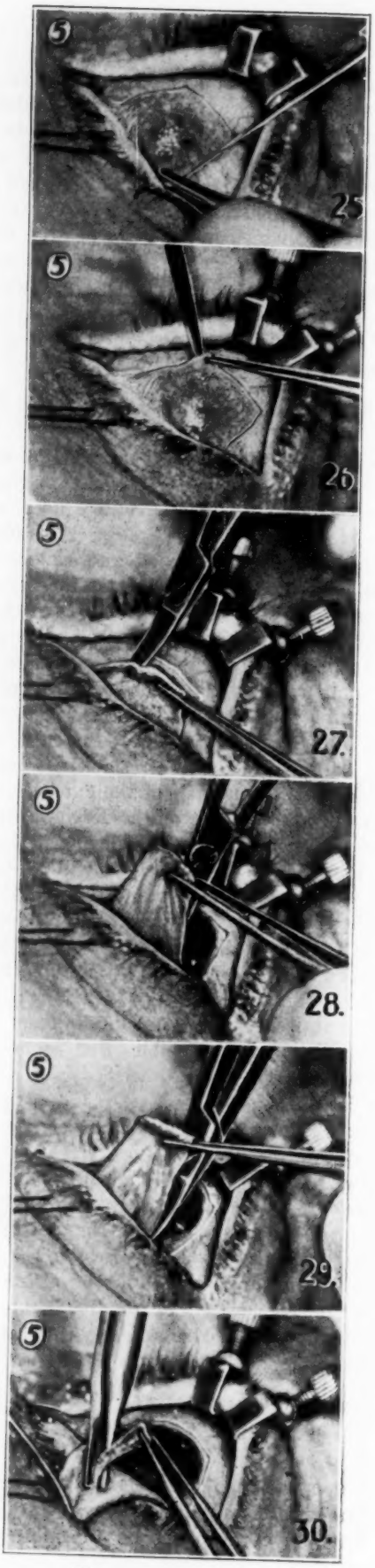


Fig. 5.—Steps 25 to 30.

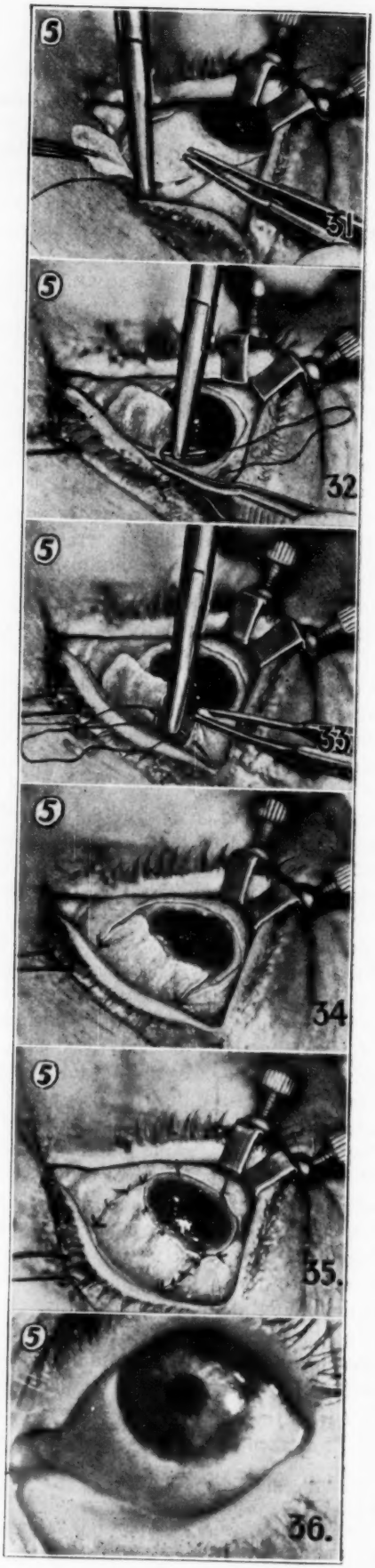


Fig. 5 (Continued).—Steps 31 to 36.

anesthesia. The procedure consists in making, with the aid of a Graefe knife, a crucial incision in the cornea extending from limbus to limbus and to a depth of approximately one-half the corneal thickness (*19*). The four corneal flaps thus limited by the crucial incision are dissected as described in previous operations. The dissection of approximately the outer half of the corneal thickness is begun with a Graefe knife, while the apex of the flap is held by the jeweler's forceps already mentioned in previous operations (*20*), and is continued with scissors, the same plane of cleavage being always maintained (*21*) until the limbus is reached. When the four corneal flaps have been dissected as far as the limbus (*22*), they are excised with scissors (*23*), the operation being finished with the application of an antiseptic ointment, such as metaphen or, better, penicillin ointment, and the application of a monocular dressing. This operation was described by Wiener in 1926. The operation as described here differs from Wiener's only in that the apex of the corneal flaps is held with a jeweler's forceps instead of the hook he advocated. Such a hook has, I believe, more tendency to tear the corneal tissue, especially when it is friable. Another modification in this technic is the use of scissors with blunt points to complete the dissection of the corneal flaps,

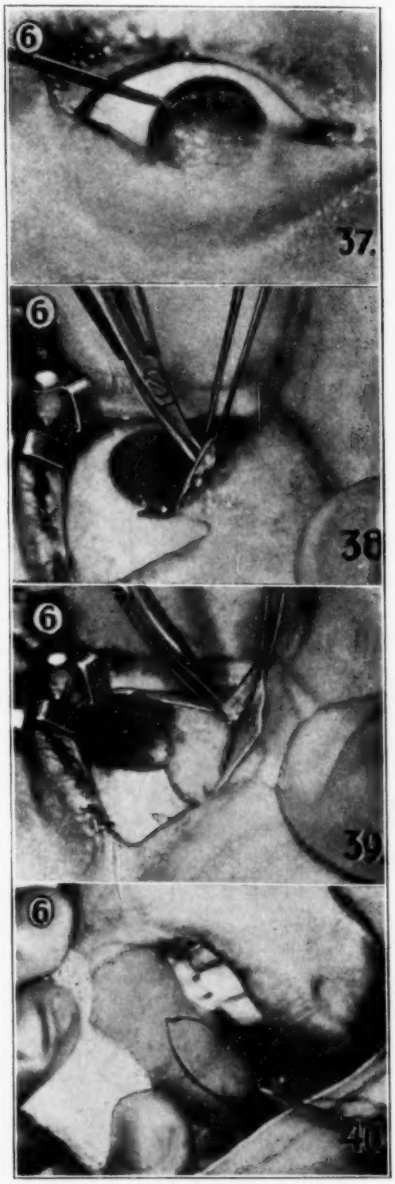


Fig. 6.—Steps 37 to 40.

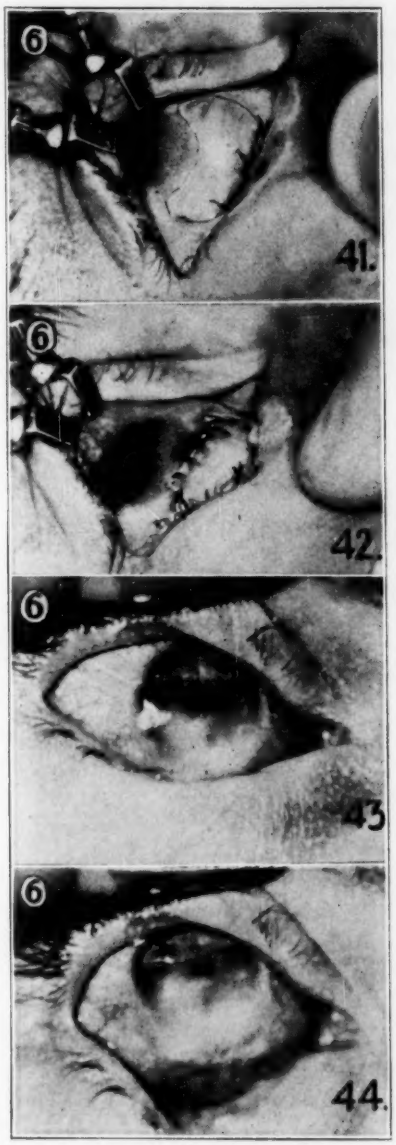


Fig. 6 (Continued).—Steps 41 to 44.

instead of employment of the knife exclusively. Those who are experienced in this type of operation will be able to dissect the corneal flaps without difficulty with the exclusive use of the knife. For surgeons with less experience, the use of scissors with blunt points will facilitate the dissection of the corneal flaps, with less danger of puncture of the inner layers of the cornea.

To prevent recurrence of corneal vascularization in these patients, I have found it useful to give treatment with roentgen rays, the irradiation starting not later than twenty-four hours after the operation.

These patients have been treated in the department of radiotherapy of the Presbyterian Hospital, under the direction of Dr. Maurice Lenz. The eye is anesthetized by the instillation of a 0.5 per cent solution of tetracaine hydrochloride. A lid speculum is inserted, and the treatment is given directly to the open eye. A voltage of 100 kilovolts, with no filter, and a target skin distance of either 6 or 9 inches (15 or 23 cm.) are used. The first treatment varies between 50 and 100 r, the dose depending on the postoperative inflammatory reaction. The following day, or two days thereafter, the interval again depending on the status of the eye, another 100 r is given. This is repeated every day or two until a total of 400 r has been given within the first week or ten days after the opera-

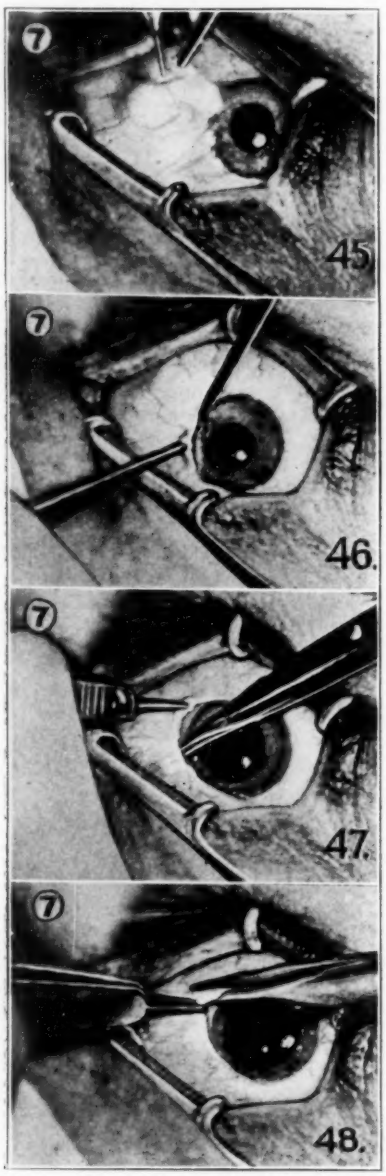


Fig. 7.—Steps 45 to 48.

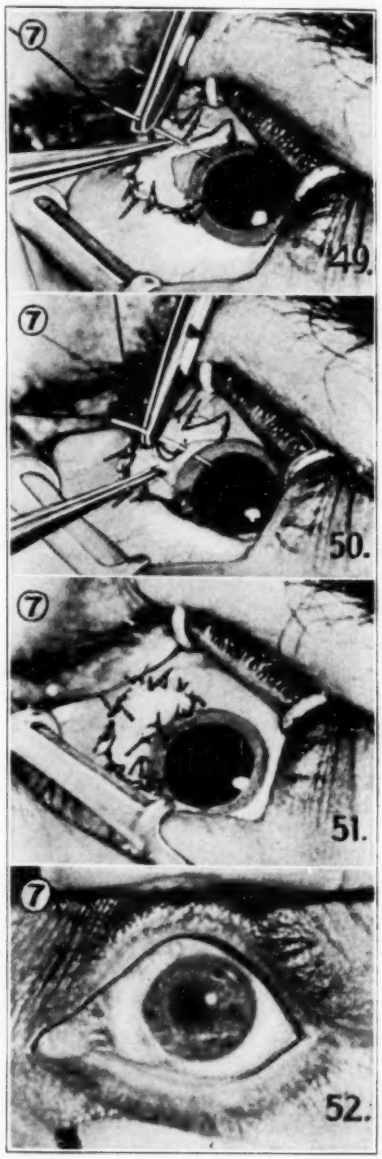


Fig. 7 (Continued).—Steps 49 to 52.

tion. In cases in which, in spite of anesthesia, the treatment through the open eye is too painful, we have given the first and second treatments with the lids closed, using 130 kilovolts, a 2 mm. aluminum filter and 100 r at each treatment. To some patients thus treated, as much as 600 r in two weeks has been administered.

Generally, ten days after the operation the cornea is fully epithelized, and at the end of one month the eye, as a rule, with little or no inflammation, appears as shown in *24*, considerably improved both in appearance and in vision. The latter may be expected to increase from light perception and projection or perception of hand movements at a distance of a few centimeters to an acuity of 10/200, 20/200 or better. We have never observed a cataract caused by these roentgen treatments.

Instead of the application of roentgen radiation to prevent recurrence of corneal vascularization, radium may be used. I have had no experience with the application of radium for this purpose, but I have seen patients treated in the department of ophthalmology of the Johns Hopkins Hospital, under the supervision of Dr. A. C. Woods. The result of radium therapy in these cases seems to be similar to that in the cases in which we used roentgen rays. I quote from the letter of Dr. Charles E. Iliff, describing the technic of radium application used at the Johns Hopkins Hospital:

"My colleagues and I use beta radiation, employing the applicator designed by Dr. Curtis Burnam, in 1925. This consists of a soda glass bulb, 0.5 mm. in diameter, containing 250 to 500 millicuries of radon encased in two small brass cylinders, which have a 4 mm. opening at one end. The outer cylinder is fastened to a 35 cm. handle for convenient application. Through the 4 mm. window all the beta radiation and most of the gamma radiation pass. The proportion of beta to gamma radiation is 100 to 1; so we usually consider the applicator with regard to beta radiation alone.

"The very soft beta rays are screened out by a heavy paper filter, or, if the lesion is superficial, unfiltered beta rays are used. The dose ranges from 1 gram for six seconds to 1 gram for twelve seconds, usually for three weekly treatments, followed by a rest period of four to six weeks. The course of therapy is then repeated until the desired effect is obtained.

"We have never had a cataract produced by radiation, nor have we been able to find a reference in the literature to one when beta radiation was used."

5. TOTAL SUPERFICIAL KERATECTOMY AND CORNEOCONJUNCTIVAL PLASTIC FOR THE TREATMENT OF VASCULARIZED LEUKOMA AND SYMBLEPHARON

This procedure is better carried out with the patient under general anesthesia because the infiltration required for local anesthesia deforms the tissues and does not permit the performance of the different operative steps as cleanly as when the tissues, without the infiltration produced by the anesthetic, have remained undisturbed. This operation is applicable to eyes with lesions similar to those described in the previous operation, but with the addition of symblepharon. In this operation the palpebral fissure must be kept open by the application of mosquito lid clamp retractors and sutures, for the symblepharon does not permit the insertion of the speculum. With the aid of a Graefe knife, a quadrangular flap is outlined by making a horizontal incision at the level of the upper limbus, followed by two vertical incisions, one starting at each end of the first incision and descending on the nasal and the temporal side tangential to the limbus to reach the borders of the symblepharon (*25*). The edge of the flap thus outlined is held with the aid of fine conjunctival forceps or jeweler's forceps, and the superficial layers of the cornea, including the vascularized scar tissue, are dissected, the dissection beginning with a Graefe knife (*26*) and then proceeding along the same plane of cleavage, with the aid of scissors with blunt points (*27*). Once the lower limit of the limbus has been reached with this dissection (*28*), the lateral incisions of the dissected corneal flap are continued temporally and nasally on each side of the symblepharon (*29*) until the lower fornix is reached. The dissection just described had the double purpose of uncovering the transparent corneal tissue (thus rendering the eye useful for visual purposes) and of making a corneoconjunctival flap, which is to be used to complete the operation as follows: The corneoconjunctival flap is thinned with the aid of scissors on its bleeding under surface, with the purpose of eliminating most of the connective tissue (*30*), which, if left, would have a tendency to cause contraction and thickening of the flap. The defect left in both the eyeball and the inner aspect of the lid is covered by means of the corneoconjunctival flap. A double-armed 0000 silk suture mounted in an atraumatic needle is used to fix to the sclera,

at the point where the fornix is to be made (*31*), both the edge of the flap (*32*) and the edge of the conjunctiva (*33*) temporally and nasally (*34*). The rest of the border of the corneoconjunctival flap is sutured to the adjacent conjunctiva and to the limbus (*35*) by means of 000000 silk mounted in an atraumatic needle, care being taken to anchor all the stitches to the sclera, as well as to the limbus, to prevent cicatricial retractions and the possible recurrence of the symblepharon, which almost invariably would take place if the stitches were placed between the corneoconjunctival flap and the conjunctiva without scleral anchorage. The rest of the conjunctiva is anchored to the sclera around the limbus to prevent the conjunctiva from slipping over the cornea and cicatrizing in a faulty position over the cornea. The operation is finished with the application of an antiseptic ointment or, better, penicillin ointment. A monocular dressing is applied with moderate pressure to prevent the accumulation of blood and exudates under the flap. The postoperative treatment also includes the application of roentgen rays, as described in previous operations, to prevent the recurrence of corneal vascularization, and the daily application of an antiseptic or penicillin ointment until complete epithelization of the cornea has taken place. About ten days after the operation, the stitches which are still present in the eye are removed, with the use of local anesthesia. If no complications arise, about one month after the operation the eye shows slight reaction, if any (*36*), with complete correction of the symblepharon, so that the appearance of the eye, as well as the visual acuity, is considerably improved. Improvement of vision may be expected to be approximately the same as that obtained by the two previous surgical procedures. The same operation described for the correction of vascularized leukoma and symblepharon of the lower lid can be used for the upper lid.

6. PARTIAL SUPERFICIAL KERATECTOMY, CORNEOCONJUNCTIVOPLASTY AND GRAFT OF BUCCAL MUCOUS MEMBRANE FOR THE TREATMENT OF VASCULARIZED LEUKOMA AND SYMBLEPHARON

Occasionally, in cases of severe symblepharon, covering a large part of the whole cornea, the tissue dissected from the cornea does not suffice to cover both the defect left in the eyeball and the inner surface of the lid. In this instance the following technic may be used:

The operation should be performed with the patient under general anesthesia, for the reasons given for the operation previously described. With the aid of a Graefe knife (*37*), an incision is made in clear corneal tissue above the symblepharon to outline the upper limit of the corneal flap which is to be dissected. While the lip of the incision is held with fine conjunctival forceps or jeweler's forceps, approximately the external half of the cornea containing the symblepharon is dissected. The dissection started with the knife is completed down to the limbus with scissors (*38*). With the aid of scissors, incisions are made along the borders of the symblepharon, down to the lower fornix (*39*). The corneoconjunctival flap thus obtained is thinned by removing from its bleeding under surface as much as possible of the connective tissue. The corneoconjunctival flap thus obtained does not suffice to cover both the defect left on the eyeball and the defect in the inner aspect of the lid. The inner aspect of the lid is covered by the corneoconjunctival flap. The defect on the eyeball is covered with a graft of buccal mucous membrane obtained with the aid of a Graefe knife from the inner aspect of the lower lip (*40*). The incision made in the mouth is closed with interrupted sutures of 0000 silk. The graft of buccal mucous membrane must be thinned as much as possible in order to eliminate the connective tissue which tends to contract. The thinner the graft of buccal mucosa the more elastic it is, adapting itself better to the defect to be covered. Also, the thinner the graft the better is the cosmetic appearance. A thick graft has more tendency not only to contract but to remain thick and unusually red. The flap of buccal mucosa is placed on the eyeball to cover the defect. The edges of the buccal mucous membrane graft, as well as the corneoconjunctival flap, are fixed to the sclera (*41*) by means of three sutures placed where the lower fornix is to be located. The remaining margins of the flap of buccal mucosa are sutured to the adjacent conjunctiva and to the limbus by means of 000000 silk, the stitches being anchored to the sclera (*42*) to prevent cicatricial retraction and the possible recurrence of the symblepharon.

The operation ends with the application of an antiseptic or penicillin ointment. A monocular dressing is applied with moderate pressure to prevent the accumulation of blood and exudates under the graft and flap. The postoperative course is similar to that described for the previous operations, with the application of roentgen rays to prevent the recurrence of corneal vascularization. At the end of several weeks, the eye which was used to illustrate this operation showed a fairly transparent cornea in the pupillary area

(*43*), with considerable improvement in appearance and slight improvement in visual acuity. The symblepharon was totally corrected (*44*). Cilia were restored to the border of the lower lid afterward by transplantation of a strip of brow to the lid. The corneal scar in the pupillary area was later successfully treated by corneal transplantation.

7. PARTIAL SUPERFICIAL KERATECTOMY AND GRAFT OF BUCCAL MUCOUS MEMBRANE FOR THE TREATMENT OF RECURRENT PTERYGIUM

This operation may be performed with local anesthesia. Once the eye is anesthetized, a subconjunctival injection of a 2 per cent solution of cocaine with a 1:1,000 dilution of epinephrine hydrochloride is made beneath the pterygium (*45*). The superficial layers of the cornea, including the apex of the pterygium, are dissected from the cornea with the aid of a Graefe knife and jeweler's forceps (*46*). When the limbus is reached, the conjunctiva, which is generally solidly attached to the eyeball, is undermined with the aid of scissors (*47*). The edge of the pterygium containing the corneal tissue is excised (*48*), together with the excess of connective tissue which remains adherent to the pterygium after it has been separated from the eyeball. The defect left in the eyeball is covered with a graft of buccal mucous membrane obtained in the same fashion as that described for the preceding operation. The graft of buccal mucosa is sutured to the adjacent conjunctiva (*49*) and at the limbus (*50, 51*), the stitches being anchored to the sclera to prevent cicatricial retraction, as well as to avert the healing of the implant in a faulty position over the cornea. The operation ends with the application of an antiseptic or a penicillin ointment. A dressing is applied with moderate pressure to prevent the accumulation of blood or exudates under the implant. The postoperative course is like that in the cases previously described, the application of roentgen radiation being necessary to prevent the recurrence of corneal vascularization or the recurrence of the pterygium. Illustration *52* shows the eye two years after the operation, with no tendency to recurrence. The technic just described was advocated by H. Gifford,[40] in 1909, and by Duverger and Velter,[41] in 1926. These authors did not suture the grafts of buccal mucous membrane to the sclera, and by this omission failed to prevent contraction of the graft.

40. Gifford, H.: The Treatment of Recurrent Pterygium, Ophth. Rec. **18**:1-8, 1909.

41. Duverger and Velter: Thérapeutique chirurgicale ophthalmologique, Paris, Masson & Cie, 1926, pp. 124-128.

SUMMARY

Superficial keratectomy in selected cases offers the ideal method of attempting to improve the visual acuity of the affected eyes. Penicillin ointment has been found to shorten the period of healing and reduce the occurrence of infection.

Dr. Maurice Lenz cooperated in the treatment of some of the patients, and Mr. Gustav Bethke helped in the preparation of the illustrations.

635 West One Hundred Sixty-Fifth Street.

DISCUSSION

Dr. F. H. Verhoeff, Boston: Dr. Castroviejo's use of the symblepharon tissue to cover the defect in the lid is ingenious, and it is evident that his early postoperative use of roentgen irradiation was essential to his success in the cases he reports.

Dr. Wendell L. Hughes, Hempstead, N. Y.: What was the final vision in the first case of keratitis, and was any radiation used? Did the ophthalmometric reading show any irregularity in the surface?

Dr. E. V. L. Brown, Chicago: What pathologic change was noted in the tissue removed in case of band keratitis?

Dr. Ramón Castroviejo: Keratectomy, meaning the excision of external layers of the cornea, is not a new procedure in cases of corneal opacities. More than one hundred and fifty years ago Scarpa described this operation in discussing corneal opacities, which he classified as nebula, albugo and leukoma. Superficial keratectomy as described in records of the past thirty or forty years remarkably resembles the surgical procedures described more than one hundred and fifty years ago. Wiener's technic of total superficial keratectomy would seem to produce irregularity of the pupillary area, and, therefore, visual acuity would not improve as much as might be expected. However, if the dissection is carried out carefully, evenness in the pupillary area can be obtained, with considerable subsequent improvement of vision.

In the case of band keratitis reported, visual acuity after the operation was 20/200. The patient had had severe iridocyclitis in both eyes. Pigment was scattered throughout the anterior surface of the lens, in sufficient amount to cause great impairment of vision. Shortly after the operation astigmatism was pronounced, but two years later the astigmatism had improved con-

siderably. The excised cornea in this case was sent to the laboratory for study, but since at that time I was concerned chiefly with improvement of the patient's vision, I did not study the pathologic report, which probably indicated the usual changes in cases of this type.

If corneal opacities still remain after the first dissection, a second dissection can be carried out to remove another portion of the external layers of the cornea, the procedure being repeated if necessary a third time, provided that the thickness of the cornea permits it. It must be kept in mind that future operations may be necessary on the cornea under treatment, and therefore it is desirable to preserve as much as possible of the corneal thickness. If the opacity invades only the outer third of the cornea only this portion should be excised.

TREATMENT OF GLAUCOMA

PAUL A. CHANDLER, M.D.
BOSTON

Many methods have been evolved for the treatment of glaucoma, but there is considerable difference of opinion as to their selection and application. In this paper I shall briefly describe the procedures that my own experience has led me to employ in treating glaucoma of various types. The conclusions are drawn entirely from personal experience and are presented to invite discussion and comparison with the experience of others.

ACUTE GLAUCOMA

Acute glaucoma may occur in some instances with practically no pain or congestion, whereas in others the pain may be violent and severe, with extreme congestion of the eye and even chemosis. Likewise, in instances of chronic glaucoma there may be congestion of various degrees. For purposes of treatment the two types—congestive and noncongestive—need not be differentiated.

Primary acute glaucoma, though much less common than the chronic form, is often so fulminating as to create one of the gravest ophthalmic emergencies. It is a distinct disease entity. The one characteristic which is invariably present is a shallow anterior chamber. If the chamber is normal or deep, one is almost certainly not dealing with primary acute glaucoma. Hemorrhagic glaucoma is sometimes first manifested as an acute attack, as is glaucoma rubrum. However, in these conditions the pupil is not widely dilated, the anterior chamber is usually normal or deep, the iris is greatly congested and a history of previous great loss of vision in the affected eye can usually be obtained. Exfoliation of the capsule of the lens may sometimes produce the picture of acute glaucoma with a deep chamber, as may a hypermature cataract or a subluxated lens. There are some eyes, apparently with a low margin of safety, in which iritis or uveitis may precipitate acute glaucoma. The differentiation of primary from secondary acute glaucoma is not always easy, because of the presence of severe corneal edema or bullous keratitis, yet it is of great importance to make it, so that the initial treatment, medical or surgical, may be the proper one. Recently Dr. David G. Cogan, director of the Howe Laboratory of Ophthalmology at the Massachusetts Eye and Ear Infirmary, demonstrated the dramatic clearing of an edematous cornea by the instillation of 1 drop of ordinary glycerin. Practically all corneal edema except that with bullae will clear in less than ten seconds sufficiently so that one can study the back of the cornea, the aqueous and the iris with the slit lamp, and if the lens is reasonably clear one can usually see the fundus. It is almost the rule to find a few cells in the aqueous in acute congestive glaucoma. In many instances a great many cells are present, and there may even be pigmented synechias. Nevertheless, if the other classic signs and symptoms are present—shallow anterior chamber, semidilated pupil and acute fulminating onset—one is dealing with primary glaucoma and the eye should be treated accordingly. If, on the other hand, the anterior chamber is normal or deep, if there are extensive posterior synechias and especially if there are precipitates on the back of the cornea, the condition is surely secondary glaucoma. If there is cupping of the disk, the condition must be treated as chronic glaucoma and only a filtering operation will suffice. If the central vein is obstructed, the condition is hemorrhagic glaucoma and the situation is hopeless, except that in a few instances the eye may be saved by cyclodiathermy. If the patient is diabetic and there is evident diabetic retinitis and dilatation of the vessels on the iris, a diagnosis of glaucoma rubrum is justified and the treatment is the same as that for hemorrhagic glaucoma.

If the condition is primary acute glaucoma, one must decide whether or not to operate and when to operate and one must choose the type of operation most likely to bring about permanent control of the tension. The important considerations are the duration of the attack and the visual acuity of the eye. Eyes vary tremendously in their vulnerability to high tension. Some eyes will tolerate high tension for days with relatively little damage to vision, while in others the visual acuity will go down in a few hours to an extremely low level. It is always preferable to lower the tension with a miotic before operating, if this is possible. The miotic

Read before the Section on Ophthalmology of the Michigan State Medical Society, October 1942, and before the Section on Ophthalmology, New York Academy of Medicine, Jan. 17, 1944.

may be given every ten minutes for two hours, then every half-hour for two hours and every hour thereafter as long as it is used.

How long should one try to reduce the tension by the use of miotics before resorting to surgical intervention? The factors which aid in this decision are the duration of the attack, the visual acuity of the eye and whether or not there is a substantial drop in the tension after two hours of miotic therapy. If the attack has been going on more than twenty-four hours, one should not defer operation more than five or six hours unless the tension is definitely falling. If the attack has been going on only a few hours, one may try miotics for as long as twelve hours if the visual acuity of the eye remains relatively good. No matter what the duration of the attack, if the vision is down to counting fingers at a few feet or less, surgical intervention should not be long delayed. In the treatment of a vulnerable eye in which the visual acuity has fallen to recognition of hand movements or less in a few hours, one should operate at once, without trying miotics at all. Five per cent neostigmine bromide combined with 20 per cent mecholyl chloride is probably the most effective miotic. One may use 4 per cent pilocarpine nitrate or 1 per cent physostigmine salicylate, or one may combine these drugs in various ways.

Assuming that the tension has been brought to normal with miotics, what further treatment should be employed? I believe that a single attack of acute glaucoma, except in extremely old people, calls for operation. Without operation almost invariably other attacks occur in spite of the regular use of a miotic, each taking some toll of the vision, until a final intractable attack occurs which may end disastrously for the eye in spite of an operation then performed. If one looks over hospital records of patients with acute glaucoma treated conservatively, one will often find a series of admissions before the final attack at which time even operation failed and all vision was lost. Acute glaucoma in old people may be treated more conservatively, for in these patients probably different factors are at work. If the tension is promptly lowered to normal with miotics, the regular use of fairly weak miotics will in most cases prevent further attacks. Even in old patients operation would be the treatment of choice were it not for its influence on the development of opacities in the lens.

If an operation is decided on, what is the operation of choice and when should it be done? If the tension cannot be brought down with miotics, paracentesis or posterior sclerotomy may be done as a preliminary measure. I seldom employ them. Of course iridectomy is the first choice, but it must be realized that iridectomy, no matter how well performed, will not as a rule be sufficient in the treatment of acute glaucoma which has been neglected or wrongly diagnosed and treated with atropine. My own experience has led me to adopt the following general rules, though I fully realize that there are exceptions to these, as to all rules.

1. If the attack is less than forty-eight hours old, iridectomy is chosen whether or not the tension comes to normal preoperatively with miotics.

2. If the attack is forty-eight hours old or more, iridectomy is done if the tension comes fully to normal preoperatively and can be maintained there with reasonable use of miotics. In any case in which the tension can be brought to normal with miotics, surgical intervention is delayed while the use of miotics is continued until the eye is relatively free from congestion.

3. If the attack is forty-eight hours old or more and the tension cannot be brought to normal with miotics or, if brought there, cannot be maintained at the normal level without vigorous use of strong miotics, a filtering operation is chosen. Under these conditions it has been my experience that iridectomy only succeeds in changing the condition from acute to chronic glaucoma. When the eye quiets down after iridectomy, too often one finds the tension running from 30 to 40 mm. (Schiøtz). Trephining as the first operation for acute glaucoma has not shown good results as a rule. In the first place, with the tension high and the anterior chamber shallow there is always the danger of the lens coming forward and blocking the opening. Then, too often, a trephine opening in a congested eye closes within a short time. The operation most successful in my hands for glaucoma of this type is sclerectomy combined with iridencleisis. I would choose this first as the operation for neglected acute glaucoma.

It must be realized that even in the instances in which iridectomy alone is clearly indicated and properly performed there may occasionally be subsequent attacks of typical acute glaucoma, particularly if the original attack was noncongestive, which are disconcerting to both surgeon and patient. Fortunately these instances are rare, and subsequent attacks are usually controlled by continuous miotic therapy.

A few points about the technic of iridectomy are of importance. I used to think that any kind of iridectomy would suffice for the treatment of acute glaucoma, but experience has convinced me that success or failure may depend on the technic of the operation.

I remember in particular 2 cases of bilateral acute glaucoma, with the condition identical in the two eyes in each case. A properly performed iridectomy permanently relieved the tension in one eye, and a poor iridectomy done at the same time on the fellow eye failed to relieve the tension. The iridectomy should be broad and basal.

For anesthesia I usually make an orbital injection of 2 per cent solution of procaine hydrochloride with epinephrine (1:40,000) in the four quadrants, 1.5 to 2 cc. in all, and wait ten minutes. If a general anesthetic seems advisable, pentothal sodium administered intravenously is satisfactory. I prefer a small sharp knife to a keratome. The incision is made without preparing a flap, since the latter is easily made with the knife. The incision made with a knife is finished more at right angles with the sclera than is an incision made with a keratome. There is therefore a tendency for the wound to gape, and the scleral bleeding, which is the most serious, always comes outside the eye. After the incision is finished, I wait without sponging until all bleeding has stopped before doing the iridectomy. This sometimes requires a considerable degree of patience, but it is well worth it, for then the only bleeding into the anterior chamber is that from the iris, which is never serious.

In treating a patient with acute glaucoma in one eye, one must be mindful of the danger of an acute attack in the other eye. Miotics should be used vigorously in the unaffected eye during the stay in the hospital. In fact, since acute glaucoma is apt to develop in the unaffected eye sooner or later, it is wise to continue indefinitely to use a miotic in it.

CHRONIC GLAUCOMA

When one first examines a patient who has chronic simple glaucoma, one measures the visual acuity and the tension and maps the visual field. These measurements indicate the status of the disease. One must then lay plans to maintain the patient's vision as long as he lives. Treatment will consist of the use of miotics or surgical intervention or both. Whatever the treatment, it must effectively prevent further reduction of the visual field. Unless the treatment accomplishes this, the diagnosis might just as well not have been made in the first place. To diagnose glaucoma and feel that one has done well in routinely prescribing 1 per cent pilocarpine nitrate to be used two or three times a day without ascertaining the effectiveness of this treatment is not to merit the confidence of the patient, to say the least. As the patient is observed over the weeks and months while receiving various treatments, one gradually gains more knowledge of his particular condition and is able to form a better judgment as to the treatment and prognosis. I have found that it is often helpful at the outset to make tension studies over a period of three or four days. The patient is hospitalized, and the tension is taken four times daily, at approximately 8 a. m., 12 noon, 5 p. m. and 10 p. m.

The first day there is no treatment. The second day 1 per cent pilocarpine is used and the third day 2 per cent. In some cases the study is continued a day or two longer and 4 per cent pilocarpine nitrate or some other strong miotic is used. Such a study will often give an idea as to the type of glaucoma with which one is dealing, and one can use at the beginning of treatment the miotic which promises to be most effective in controlling the disease. The study may indicate at the outset that an operation must be done in order to hold the tension at reasonable levels. In such studies one observes several types of tension curves. There is a type in which the tension in the untreated eye never goes above the low twenties (fig. 1). Miotics have no significant effect on tension in this type. There is a type in which the tension is usually low but is definitely elevated at some one period of the day. Miotics are often extremely effective against this type of glaucoma by eliminating the periodic elevations which are causing damage to the eye (fig. 2). Then there is a type of glaucoma with relatively high tension in which strong miotics are usually required to maintain the tension at normal levels (fig. 3), and another type with high tension in which even the strongest miotics are not effective (fig. 4). Relatively advanced, so-called capsular glaucoma is usually characterized by high tension of the latter type.

In making the often difficult decision as to whether or not to operate such studies of tension are often exceedingly helpful. There are many other factors to be considered however. The behavior of the visual field over a period of time is, of course, the most important factor in the decision. The general health of the patient and his faithfulness in carrying out the recommended treatment must be considered. One must have regard as to whether the patient is easy-going and placid or a tense, nervous person on whom life imposes many stresses and strains. All other things being equal, one would be more inclined to operate early on persons of the latter than on persons of the former type. Perhaps the most important single factor is the age of the patient. I believe that few patients with an average life expectancy of twenty or thirty years can have glaucoma held in check over such a long period with miotics alone and therefore that

practically all patients under 60 years of age in good general health should have an operation. For patients between 60 and 70 years of age, I believe an operation is indicated if extremely strong miotics are required to hold the tension at normal levels, for in such instances sooner or later miotics will no longer be effective. It is in the treatment of patients over the age of 70 that it is often most difficult to decide the question of the desirability of surgical intervention. In treating such a patient, if the diagnosis is established early and there is a good visual field, one may be content to allow a slow loss under miotic therapy, whereas if the disease is already far advanced when the diagnosis is made, surgical intervention may be indicated at the outset, for the patient cannot afford to lose even a little. In older patients I feel that a Schiøtz tonometer reading in the thirties is an indication for operation, especially if there is rapid reduc-

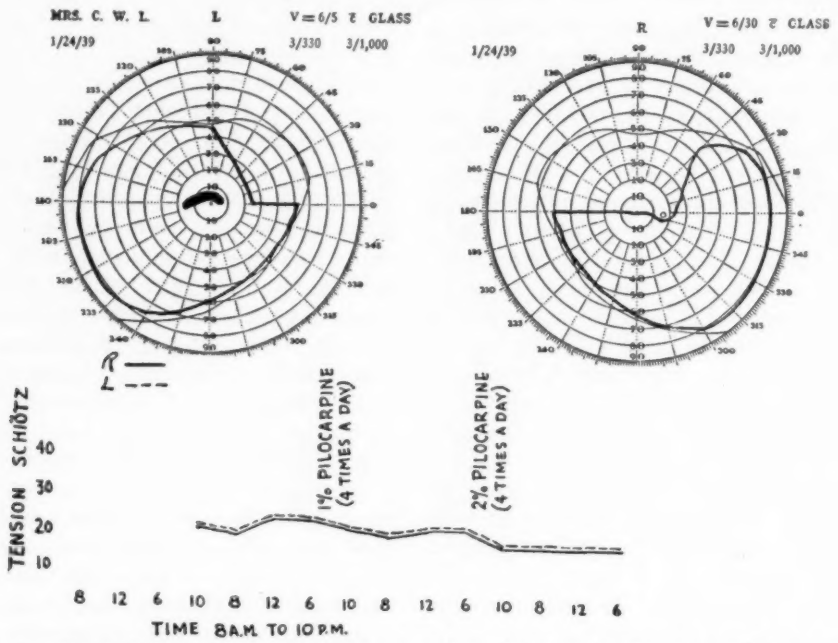


Fig. 1.—Visual fields and tension curve for a patient with chronic glaucoma with low tension.

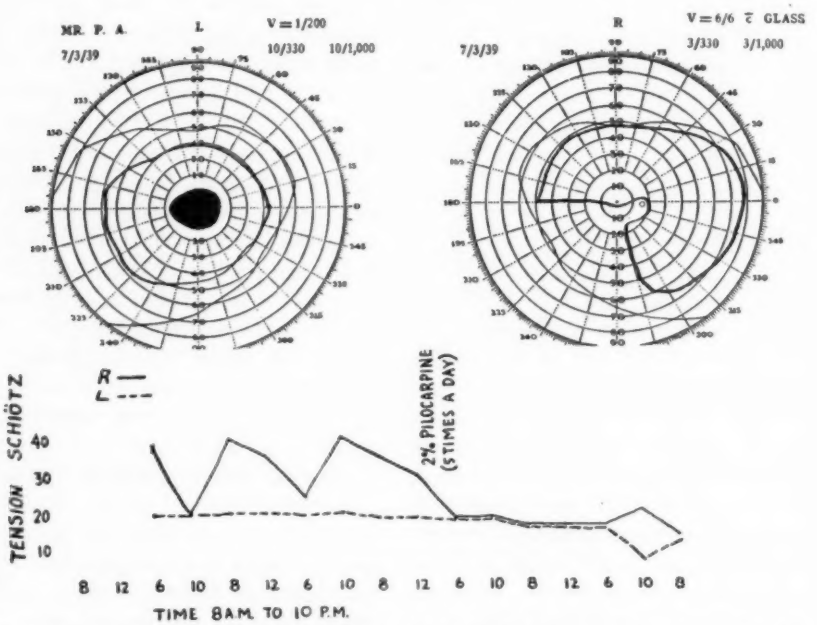


Fig. 2.—Visual fields and tension curve for a patient with chronic glaucoma with generally low tension elevated at one period of the day.

tion of the field. In all older patients, the indications for operation must be relatively stronger than in younger patients for any surgical procedure to relieve glaucoma in these patients almost invariably causes the development of opacities in the lens, or causes a definite increase in opacities already present. It should be emphasized that in cases in which for one reason or another one wishes to avoid surgical intervention stronger miotics can be used at frequent intervals. When a 1 or 2 per cent solution of pilocarpine nitrate used three or four times a day is ineffectual, a 4 per cent solution can be used as often as every two hours during the day.

As to the type of operation to be employed each one of us must be guided by his own experience. Let us employ the operation that we can perform most skillfully and that gives us the best results. I favor trephining for most younger patients. In my hands it is the most successful operation for holding down the tension for a long period. I think it is particularly effective for eyes with relatively low tension. The 1.5 mm. trephine is used routinely, but in the treatment of juvenile glaucoma the 2 mm. trephine is always used. I definitely favor iridencleisis for all older patients, for I find that it seldom results in the hypotony so frequently seen after trephining under such conditions, and this means that it

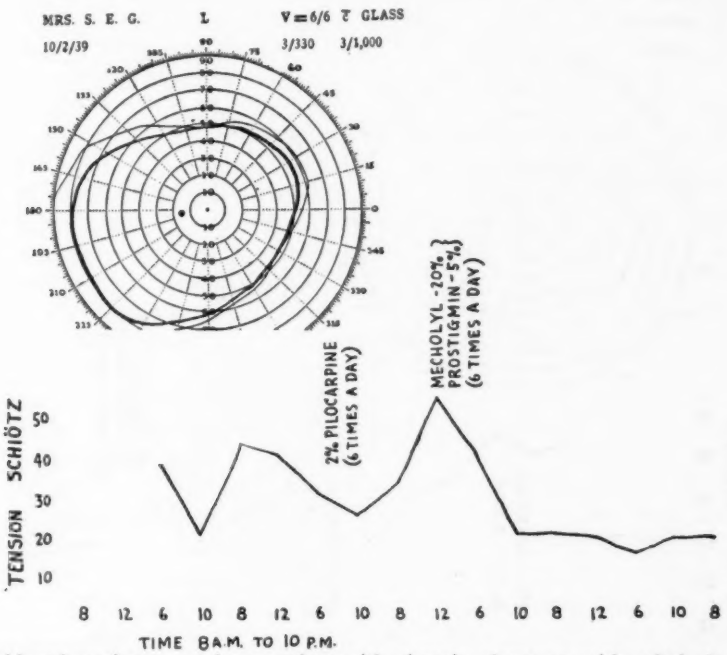


Fig. 3.—Visual field and tension curve for a patient with chronic glaucoma with relatively high tension which is controlled by strong miotics.

In patients in the low tension group, in whom the tension is always under 25 mm. (Schiøtz), miotics have little or no effect, but the loss of field is relatively slow. I believe, therefore, that a patient 50 years of age with glaucoma of this type should have an operation but that for a patient of 65 or over an operation is practically never indicated.

It is impossible to make fixed rules in regard to surgical intervention in chronic glaucoma, but in general it may be said that most of us have had many more occasions to regret not having resorted to an operation than we have had to regret operating. Another fact to be remembered is that eyes with early glaucoma as a rule do much better after surgical intervention than those in which the disease is advanced. is much less apt to cause cataract. In general, I favor an iris inclusion operation for eyes which must be operated on in the presence of a relatively high tension. Under these conditions after a well executed trephination I have seen the lens come forward and block the opening, completely nullifying the operation. Iridencleisis combined with sclerectomy is my operation of choice for congested eyes, as it offers the best chance for permanent filtration. Also, when I perform an iris inclusion operation for a younger patient, a sclerectomy is done. I treat glaucoma due to exfoliation of the capsule of the lens no differently from glaucoma in which exfoliation is not present. I believe that the iris inclusion operation with sclerectomy is better than trephining for such a condition, since I have several times

seen a perfectly functioning trephine opening close within a year or two after the operation.

As to the technic of the iris inclusion operation, one may of course use either a keratome or a knife for the incision. An incision made with a keratome probably offers the least trauma to the eye, but if the anterior chamber is excessively shallow it requires a good deal of skill to make it properly. One may prepare the flap beforehand as in the trephining operation, or one may make the flap with the knife, or if the keratome is used the Reese type of incision may be made. My own preference is to use the knife for the incision, as in iridectomy, without previous preparation of the flap. With a little care one can make with the knife as large a conjunctival flap as necessary. It has seemed to me that this caused less trauma to the conjunctiva than if the flap were prepared beforehand, and there is better visibility in making the incision. As in iridectomy in which the incision is made with a knife all blood from the wound invariably escapes on the outside of the eye rather than into the anterior chamber. After the incision is finished I like to wait without sponging until the scleral bleeding has stopped. If one continually sponges, the blood may be forced into the anterior chamber. The iris should be withdrawn with great care, so as to avoid unnecessary trauma to the pigment epithelium. I usually cut the sphincter on each side of the forceps. When this is done, the forceps is released and the iris is not touched again. Under certain circumstances a tongue of iris is prepared and the sphincter is left intact. No attempt is made to rearrange or flatten the tongue of iris. Any such manipulation may further traumatize the pigment epithelium, which must remain more or less intact in order that a proper filtering scar may develop. Atropine is used postoperatively, not as a routine but according to the amount of reaction, the size of the pupil and the amount of blood in the anterior chamber. In some instances no atropine is used at all. When sclerectomy is combined with iris inclusion, after the incision is finished the conjunctiva is separated from the anterior scleral lip by gentle dissection with the tip of a keratome and then a piece of the sclera is bitten off with a Holth or a Berens punch.

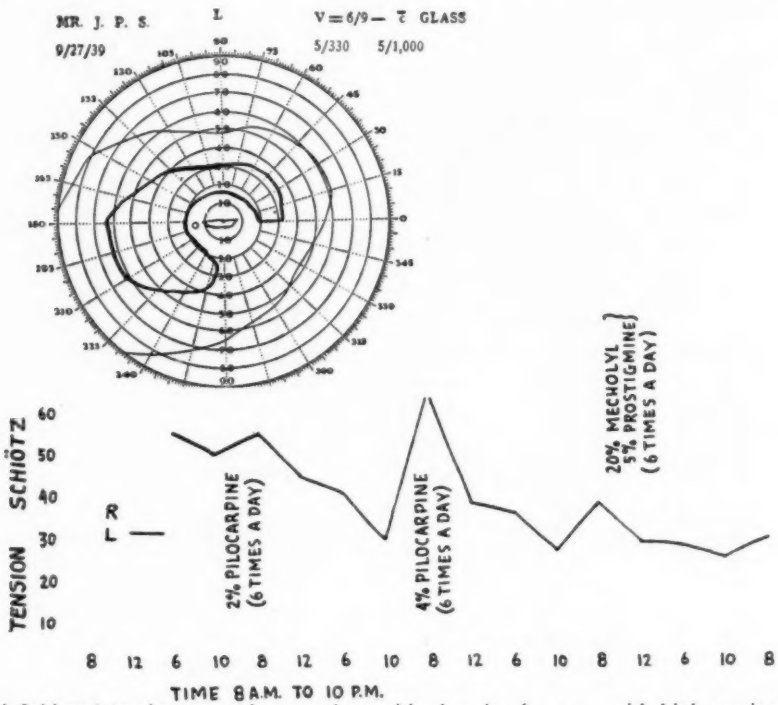


Fig. 4.—Visual field and tension curve for a patient with chronic glaucoma with high tension which is not controlled by strong miotics.

Chronic glaucoma coexisting with immature cataract is a particular problem. Treatment depends on the extent of the cataract and on the severity of the glaucoma. Miotic therapy, if it is reasonably effective in controlling the glaucoma, is always preferable until the cataract has advanced sufficiently to warrant its removal. Unfortunately, if the opacities in the lens are central, the miosis may diminish central vision to an extent that is incapacitating to the patient. If the vision is cut down to 20/50 or less by the opacities in the lens, intracapsular extraction with

a broad basal iridectomy will often control the glaucoma. In instances in which the tension remains too high after such an operation, miotic therapy, perhaps combined with the use of 1 or 2 per cent epinephrine, will usually be effective. In rare instances further surgical intervention, preferably cyclodialysis, will be necessary.

If central vision is 20/40 or better without miotics and miotics reduce the vision below 20/40, I believe that broad iridectomy followed by miotic therapy is preferable to immediate extraction of the cataract. Of course a filtering operation would be expected to afford better control of the tension in such cases, but if performed on an eye with already well developed opacities in the lens it almost invariably causes a rapid increase in the opacities. To be sure, if the cataract develops rapidly, extraction of the lens can be done, but the operation is undoubtedly more difficult to perform on such an eye. After iridectomy, on the other hand, if extraction of the lens becomes necessary, it is simplified by the previously performed iridectomy.

When any operation designed to produce a filtering scar is done, it is extremely important to keep the patient under close observation during the first few weeks after the operation. In some instances there is nothing to be done, but in others early "massage" [1] of the eye may make the difference between success and failure. By using the proper amount of "massage," the tension may finally become stabilized at the desired level. In some instances if one waits as long as two weeks after operation before beginning pressure on the eye it may be too late. I like to see the patient at least once a week during the first six weeks after the operation.

BUPHTHALMOS

For the treatment of buphthalmos or infantile glaucoma iridencleisis has proved more successful in my hands than trephining. In any operation for glaucoma of this type it is of the greatest importance that adequate massage be carried out by the parents after the child leaves the hospital. In fact, I believe that the after-care by the parents is so important that the operation is almost certain to fail without it. On the other hand, if firm pressure is made on the eyes four times a day, day after day without fail, one can anticipate a much higher percentage of success than is generally realized. One must take the parents into one's confidence and carefully explain the nature of the disease and the principle of the operation. They must be made to understand that their role is as important as that of the surgeon, that they must do their part if the disease is to be controlled. There is usually hypotony for one to two weeks after the operation. Pressure on the eye is begun as soon as the tension rises to normal. Barkan's operation of goniotomy for this type of glaucoma sounds promising; I have had no experience with it.

In the general management of patients with primary glaucoma, I feel it my duty to remind them by letter or telephone when it is time for them to be seen and to continue to do this until they come to the office or I am assured they are under the care of another oculist. Patients being treated with miotics in whom the tension seems to be under control are seen three times a year. After successful surgical intervention they are seen twice a year. Of course any unusual elevation of tension calls for more frequent visits until it can be ascertained that the revised treatment promises to hold the tension within normal limits.

SECONDARY GLAUCOMA

If it is difficult to lay down rules for the treatment of primary glaucoma, it is well nigh impossible to do so for the treatment of secondary glaucoma, every instance of which presents its own peculiar problem. The only general rule which can be made is that in the treatment of secondary glaucoma one should employ conservative measures as long as possible.

In the treatment of glaucoma secondary to acute iritis I always continue the use of mydriatics. The first move is to use 1 or 2 per cent solution of epinephrine bitartrate three or four times a day. If this fails, paracentesis is tried; and if paracentesis holds the tension down for only a few hours, I try a Reese incision.[2]

With uveitis, a more chronic disease, Reese incision, repeated as often as necessary, will

1. By "massage" is meant firm, steady pressure on the eyeball.

2. For combating secondary glaucoma an incision without iridectomy was introduced by Verhoeff at the Massachusetts Eye and Ear Infirmary many years ago. He termed it the "Reese incision" because it was similar to the incision made by Robert Reese in performing iridectomy. Whether Reese himself ever employed it without iridectomy, I do not know. The incision is made subconjunctivally, usually from above, with a keratome, and it produces an exceedingly small cut in the conjunctiva far from the scleral incision. Previously Verhoeff had made the same sort of incision by means of a Ziegler knife-needle. The tip of the keratome is engaged in the conjunctiva 7 or 8 mm. from the limbus and passed subconjunctivally to a point about 1.5 mm. from the limbus and then into the anterior chamber. The incision into the anterior chamber is usually 6 to 7 mm. broad. No suture is used. Pressure on the eye is begun the following day and repeated several times daily, usually by the patient himself. Each time pressure is used it must be applied continuously until the eye is soft. The incision may thus be kept draining for days, sometimes for weeks.

often tide over a difficult situation. A year or two ago, in treating a patient with bilateral uveitis with secondary glaucoma, five Reese incisions were made in each eye over a period of a few months. In the end the uveitis quieted down, the tension remained normal, the vision in each eye was 20/20 and the visual fields were full. In the treatment of a subsequent attack of uveitis lasting several months a single Reese incision in each eye was sufficient to control the tension, since the patient was by that time so well trained in massage that he never allowed the incisions to close. In the treatment of intractable secondary glaucoma, such as that in sympathetic pveitis, if the Reese incision proves ineffective I have occasionally used iridencleisis with sclerectomy successfully.

In the treatment of glaucoma due to iris bombé I favor iridectomy rather than transfixion of the iris, for the slitlike opening produced by transfixion will not infrequently become closed by hemorrhage or exudate. When iris bombé is present without increased tension, it is wise to defer operation until there is elevation of tension. In some eyes with iris bombé the tension may remain normal or even subnormal, possibly owing to atrophy of the ciliary body. Iridectomy on such eyes will often result in atrophia bulbi and should not be done unless the eye is practically blind as a result of closure of the pupil and the presence of opacities in the lens. In such circumstances iridectomy may be combined with extraction of the lens in a final attempt to obtain vision, for the situation is such that there is nothing to lose and possibly something to gain by operating.

Glaucoma following discission of the intact lens or following traumatic cataract calls for prompt linear extraction of the lens. Increased tension after incomplete removal of the lens is probably best treated by repeated paracentesis. The tension usually subsides as the lenticular matter is absorbed.

Hypermature cataract often causes acute glaucoma. Some oculists favor iridectomy in the treatment of this condition, followed by extraction of the lens at a later date. Whether or not the tension can be brought to normal with miotics preoperatively, I much prefer extraction of the lens with broad iridectomy as the first operation. I believe one runs an unnecessary additional risk in doing a preliminary iridectomy. Following preliminary iridectomy for acute glaucoma due to hypermature cataract there may be serious bleeding into the anterior chamber or persistence of a flat or shallow chamber, with return of the glaucoma and persistent congestion, making an extremely complicated situation.

After severe contusion of an eye the anterior chamber is sometimes black with blood and the pressure within the eye elevated. There is no visible structure to the clot, and the cornea resembles a section of a black marble. Since such a clot will never absorb in time to save the eye, an operation should be done at once. Aside from the possibility of damage from the glaucoma, hematogenous pigmentation of the cornea will begin sometimes within thirty-six hours. An incision should be made with a keratome and the blood clot should be washed out of the anterior chamber.

The situation is quite different, however, if one can see structure to the clot or if any portion of the iris is visible. In such conditions the blood is usually absorbed rapidly, and the eye should be left alone if the tension is normal or only slightly elevated. A considerable elevation of tension can often be successfully combated with miotics, but if it cannot be controlled paracentesis should be done. When the blood is absorbed and the tension is normal, the use of a miotic can be discontinued and a mydriatic employed for the remainder of the period of convalescence. During the first few days of treatment the use of hot compresses should be avoided, for their employment undoubtedly favors continued bleeding.

Subluxation or complete dislocation of the lens may cause glaucoma. When the lens becomes dislocated into the anterior chamber, it usually causes acute glaucoma and must be removed without delay. Increased tension due to partial subluxation of the lens backward, from trauma, sometimes proves to be transient and can be successfully controlled with miotics. If the tension is uncontrolled, however, the lens should be removed. When the lens is completely dislocated into the posterior chamber, it is sometimes well tolerated without any elevation of tension; under these circumstances it is best left alone. If intractable glaucoma ensues, however, the lens should be removed if possible. Removal of a subluxated or completely dislocated lens is a formidable procedure and should not be undertaken without strong provocation.

The glaucoma that sometimes occurs after uncomplicated extraction of a cataract and especially after needling of a secondary membrane constitutes a difficult problem. In a few instances the condition is controlled by miotics alone, and the combined use of a miotic and 1 or 2 per cent epinephrine is particularly helpful. For operative treatment I continue to employ cyclodialysis, but I must confess that in my hands it is not always effective. When cyclodialysis fails, trephining with the 2 mm. blade will not infrequently be

successful. As a last resort cyclodiathermy may be tried, but only as a last resort, for I consider it to be heroic treatment. If cyclodiathermy is extensive enough to control the tension, it sometimes leads to atrophia bulbi.

A general rule for the surgical treatment of secondary glaucoma is to use all the conservatism consistent with maintaining the vision of the eye. Never employ a big operation if a minor one may tide things over.

One grows in wisdom in treating glaucoma only by continually inquiring into the causes of failures. If one form of treatment fails to relieve glaucoma of a given type another should be tried the next time the same condition is encountered. Let us not continue to make the same mistake over and over again. The treatment of glaucoma is one of the major problems of ophthalmology and requires the employment of the ophthalmologist's best judgment and skill.

5 Bay State Road.

DISCUSSION AT MEETING OF NEW YORK ACADEMY OF MEDICINE

Dr. Arnold Knapp, New York: We know that Dr. Chandler has been particularly interested in surgical treatment of glaucoma, and we are glad that he has taken the trouble to give us the benefit of his experience and careful observation. There are a great many questions we should like to ask him, but I am going to speak of only one or two of them.

First, I was particularly interested in his being able to accomplish something in the treatment of buphthalmos by means of pressure. My results in the treatment of buphthalmos have been bad. Frequently the patients disappear from observation and no one knows what the final results are.

Second, in speaking of operations for acute glaucoma, Dr. Chandler mentioned a case in which the formation of a perfect coloboma gave a good result and a poor iridectomy failed to relieve the tension. In my experience it is not always possible to obtain a clean coloboma in an eye with acute glaucoma, because the iris is too friable, and an imperfect coloboma often gives a surprisingly good result.

In discussing surgical treatment of hypermature cataracts that cause glaucoma Dr. Chandler said that the cataract should be extracted immediately, which is an interesting observation. I have always tried to get the eye in a better condition by an iridectomy and then, after about three weeks, extracted the cataract in the capsule.

I noted that Dr. Chandler is partial to iridencleisis among the decompression operations for chronic glaucoma. This reminds me of what Dr. John Weeks always used to say: "Do whatever operation you have confidence in."

Dr. Mark J. Schoenberg, New York: We are all much obliged to Dr. Chandler for his fine paper, especially for the emphasis he placed on the importance of proper technic and for his discussion of the indications for the various operations for glaucoma. The details he mentioned concerning the after-treatment are certainly welcome, and I agree with him entirely that so-called minor details are apt to determine whether the outcome of an operation is a success or a failure.

May I mention one more point which is of equal importance? This is the avoidance of surgical trauma. It is one thing to know the technic and an entirely different thing to be gentle in assaulting the tissues with surgical implements. Much of the "roughness" of some surgeons may be due to insufficient training or to a state of tension, but some of it is due to the fact that teachers are not putting sufficient emphasis on the importance of gentleness in handling the tissues.

If eyes come out of the operative ordeal without much inflammatory reaction, they owe this not only to the patient's vitality but often mostly to the gentleness of touch of the man who performed the operation.

Dr. John McLean, New York: I should like to thank Dr. Chandler for his practical and instructive talk and to add a few questions. I noticed that he was inclined to stress the combination of cataract and glaucoma, and I should like to ask how he handles the extraction of the cataract after a successful filtering operation. That sometimes can be an important point. The converse, which I do not think he mentioned, occurs when a typical aphakic glaucoma develops apparently as a result of an operation for cataract which has gone along rather smoothly except for delayed reformation of the anterior chamber. What is the best approach under these circumstances? Some of us are enthusiastic about cyclodialysis and believe that this is the only condition for which cyclodialysis has a real value.

Dr. Chandler spoke about the danger of expulsive hemorrhage in operations on eyes which are hard, especially in extraction of a cataract. I wonder what he thinks about the use of retrobulbar injections, particularly of sizable amounts of epinephrine, as a means of reducing the tension, possibly combined with massage before opening the eye to help reduce the possibility of hemorrhage.

Finally, I noticed that Dr. Chandler prefers an incision made with a knife in the Lagrange operation because he can make a more perpendicular cut at the limbus with a knife than with a keratome. I wonder whether he has any particular reason for not using the so-called "scratch" or ab externo incision, which can be made perpendicularly.

DR. PAUL CHANDLER, Boston: In regard to buphthalmos, which Dr. Knapp mentioned, I would say that to my knowledge I never cured a patient with that disease until the last 3 were treated by the "pressure" method. The parents of the 3 children were extremely cooperative and were well instructed beforehand. I think the eyes are still holding. The first patient has been followed for three years, the second for eighteen months and the third for nine months. The corneas have not enlarged, and the pressure has remained normal.

A poorly performed iridectomy may sometimes work better than a well performed one, especially if a tab of iris is left in the wound and a filtering scar develops.

In the treatment of hypermature cataract and acute glaucoma, I was taught to do an iridectomy. I have seen several eyes with this combination of conditions which had copious bleeding into the chamber after the iridectomy, but, although I have not encountered many of them, I have never had any difficulty in extracting the lens as the first operation.

I agree that trauma in surgical procedures should be avoided as far as possible. In doing a trephining I think it is better not even to sponge, but to wash away the blood by irrigation when splitting the cornea. In iridencleisis it is of the greatest importance that the iris be grasped with extreme gentleness when it is withdrawn. One or more cuts may be made, according to the preference of the surgeon, but then the iris should be released and not touched again. I make no attempt to flatten or smooth it, because if the pigment epithelium is damaged a filtering scar will not develop.

Extracting a lens in the presence of an adequate filtering scar is not an easy operation, particularly if there is a large bleb encroaching on the cornea. This condition sometimes occurs following trephining. Such eyes do not require any flap. I usually cut the conjunctiva loose on either side of the bleb and put in so-called corneoscleral track sutures, which under these circumstances should be largely corneal, and then make a small corneal incision. If the sutures are largely corneal it is easier to make the section than if they are placed in the usual fashion. The tracks are then rethreaded. The lens is usually tumbled, since the larger scleral lip makes it more difficult to grasp the lens above. The capsules are usually fairly strong. The corneoscleral suture gives a satisfactory closure of the wound.

I think that glaucoma in aphakic eyes is probably the most difficult to deal with. Fortunately in a few eyes with this condition the tension can be lowered by the use of miotics plus the use of 1 per cent epinephrine once or twice a day. If this fails, I choose cyclodialysis as the first operation, as Dr. McLean said he did. Either I do not do this skilfully or my luck has been bad, for I have not had the best of success with it. If this treatment fails, I like to do a 2 mm. trephining instead of repeating the cyclodialysis. Then pressure on the eye will often cause a small localized bleb to develop with a satisfactory result. It might seem that the vitreous would get into the trephine hole when this operation is done, but it is surprising how seldom that occurs.

I agree that pressure on the eye is helpful in conjunction with retrobulbar injection when the lens is extracted from a hard eye. I use retrobulbar injection in every intraocular operation, and I like to wait five or ten minutes after the injection is given before operating. In handling hard eyes I maintain firm pressure on the eye all the time I am waiting for the anesthetic to take effect. Sometimes hard eyes will soften surprisingly during that period.

INDUSTRIAL INJURIES OF THE EYE

ELBERT S. SHERMAN, M.D.

NEWARK, N. J.

The treatment, including operative procedures, of industrial injuries of the eye does not differ materially from that of nonindustrial injuries, but because of the workmen's compensation laws, certain factors other than treatment enter into their management.

Compensation for "personal injury to an employee by accident arising out of and in the course of his employment" is the phraseology used in these laws, which since 1911 have been almost universally adopted in this country. Thus, with few exceptions, every case of industrial injury is what is commonly termed a compensation case. It may also become a litigation case.

I do not want to pose as an industrial ophthalmologist, but having practiced ophthalmology for many years in the center of a large industrial area in which there is a great variety of industries, I have had a considerable number of patients with injuries of the eye sent to my office. Because of the great acceleration of industrial activity during the past two or three years, there has been a great increase in the volume of this class of work.

Employers and insurance companies have gradually learned by expensive experience that it pays to have ocular injuries, even apparently trivial ones, taken care of by an ophthalmic surgeon, preferably one familiar with compensation work. They are willing to pay well for honest, competent service. The president of one of the large insurance companies said:[1]

> The modern insurance company no longer considers it economy to organize its staff of surgeons on the basis of low fees. There is a sincere desire to give the industrially injured the highest grade of surgical care that can be secured.

Injuries to the eye are the most expensive type of industrial accidents.

Compensation includes (1) the cost of hospitalization and surgical care, (2) pay for temporary disability (loss of time) and (3) pay for permanent disability (impairment or loss of function). Permanent disability may be partial or total. In the case of an injury to the eye, after treatment has ended and a necessary period of observation has elapsed, the ophthalmic surgeon is asked to give an estimate of the permanent disability, if any. If there is no loss of visual function, or if visual acuity is reduced below the arbitrary point of industrial blindness, which is usually 20/200 but varies in different states, the estimate is easily made. However, an estimate of no permanent disability is sometimes disputed by the employee, who has now become what is known in compensation court parlance as the petitioner. By this time, and often much earlier, he has a lawyer, who hires, for a contingent fee, a more friendly expert on the eye. Then, if a satisfactory settlement cannot be effected with the employer, who is now termed the respondent, all parties must appear in the compensation court.

Computation of the percentage of loss of vision in cases of partial permanent disability is often more difficult and requires, besides good diagnostic ability, a knowledge of the standard method of evaluation of industrial visual disabilities approved a few years ago by the American Medical Association, or some modification of this method acceptable in the state in which one practices. This phase of the subject has been discussed so well and authoritatively by Snell[2] that further reference is unnecessary. Fortunately, in all but a small percentage of cases, the injury is trivial, and the question of compensation does not come up.

I am aware that compensation work is distasteful to, and is shunned by, some ophthalmologists. This is not surprising in view of newspaper reports of practices revealed in New York by the recent investigation of abuses of workmen's compensation laws, in which it was charged that 3,000 physicians received $5,000,000 in "kickbacks" last year. However, I know of no more useful or satisfactory work than the care of industrial injuries of the eye, particularly now, when the conservation of man hours and sight is so important to the war effort,

Read at a meeting of the New York Academy of Medicine, Section of Ophthalmology, Dec. 20, 1943.

1. Burns, F. H.: Paper presented at the meeting of the Board on Traumatic Surgery of the American College of Surgeons, 1928.

2. Snell, A. C., Sr.: A Treatise on Medicolegal Ophthalmology, St. Louis, C. V. Mosby Company, 1940.

and it is regrettable that some good ophthalmologists fear to risk the smearing of their reputations by taking cases of this kind. In New Jersey, I believe, there are few such abuses, at least among the ophthalmologists. There is no reason that industrial injuries of the eye cannot be handled as honestly as any other kind.

Another feature of industrial work that is objectionable to some physicians is the necessity of making reports. When an employee is disabled by injury, the employer or his insurance carrier pays him a fixed percentage of his wages while he is unable to work. If there is some degree of permanent disability, the payments are continued for a specified time, the length of which depends on the nature and extent of the disability. In making these payments and in determining when they shall cease, the insurance companies depend almost entirely on the reports of the surgeon, and in the absence of this authority payment to the injured workman is often delayed. The Workmen's Compensation Bureau will not make any award for permanent disability without a physician's report. Reports are, therefore, essential. If one has a considerable number of cases of industrial ocular injuries, the work can readily be organized so that the reports are made quickly and almost automatically. My associates and I have most of them taken care of by the nurse or her assistant. We have found it necessary, for several reasons, to have waiting and treatment rooms separate from those used for other patients. To physicians who are not familiar with them, the usual forms for report of accidents used by the casualty companies may look formidable, but for slight injuries of the eye, which include the large majority of accidents involving the eye, only three or four essential questions need be answered. Several years ago I made a short form for the report of injuries of the eye, which has been a great time saver. On it are spaces for the name of the workman, whether he is to return for further treatment, the diagnosis and an estimate (actual or tentative) of temporary or permanent disability. For most cases this gives all the information needed or desired. It is given to the workman in a sealed envelope to be taken to his employer, who, if he is not self insured, forwards it to his insurance carrier with his accident report.

When there is likely to be permanent disability, a final report should not be made until function has been restored as fully as possible. In most cases it is evident at once that there will be no disability. In a few cases it can be seen, from the nature and extent of the injury, that total loss of vision is certain. In other cases, after the acute symptoms have subsided, sufficient time should elapse for the gradual improvement which often occurs, and may continue over a period of months, or for the development of sequelae, before a final report is made. This applies particularly to corneal injuries; uveitis; opacities in the lens and vitreous; partial optic nerve atrophy; paralysis of the ocular muscles, both external and internal, and certain lesions of the fundus. While one should be cautious in making a prognosis, astonishing improvement often occurs in the impairment of vision caused by serpiginous and other traumatic ulcers, or in the astigmatism resulting from nonpenetrating wounds of the cornea and some other conditions. This is also true of traumatic paralyses of the extraocular muscles. The diplopia often clears up completely in from six months to a year. Fortunately, in compensation work, the surgeon may usually keep the patient under observation as long as necessary. He should not permit himself to be hurried into making a final report prematurely. The period of observation may sometimes last for many months after the patient has returned to work.

In industrial work an accurate history of the accident is important. This may be brief or more extended, depending on the nature of the injury. Most industrial injuries of the eye are trivial and recover with no permanent impairment of vision or loss of time, but every case has the elements of a potential claim. Therefore, when the patient is first seen, as a matter of justice both to the employer and to the employee, a record should be made of when, where and how the accident occurred; of previous injuries; of the vision of both eyes, and of any preexisting pathologic changes, such as scars on the cornea or opacities of the lens. Not infrequently, sometimes months after a slight injury, an employee will, often in good faith, falsely attribute to it a serious loss of vision, which is really due to a preexisting condition. It has been shown that such defects are present in 20 per cent of workmen.[3] Unjust claims of this kind cost insurance companies and employers large sums of money. If an adequate examination and record have not been made, it is sometimes impossible to defeat such a claim. Because of the nature of the case, it is not always possible to make an accurate visual test of a recently injured eye, but this should be done as soon as conditions permit. Simulated amblyopia seldom appears until the employee has had time to reflect on the possibilities of capitalizing

3. McAuliff, G. R.: Noncompensable Visual Defects in Industrial Ophthalmology, Am. J. Ophth. **11**:714 (Sept.) 1928.

on his accident. If the impairment of vision is not satisfactorily accounted for by the recent injury, the real cause should be ascertained.

Several years ago, Zimmer,[4] director of the Division of Workmen's Compensation, Department of Labor of New York, in a paper read before this section, commented on the importance of records. He said:

> The department is constantly pointing out to doctors the necessity of making and retaining complete case records. Experience has taught that record keeping is even more necessary in eye cases than in most other forms of injury. . . . Many physicians seem reluctant to answer the all-important question as to whether the injury has produced loss of vision. . . . It should hardly be necessary to point out that the reports should include visual tests and a pathologic description of the uninjured eye. These data are of grave importance in many cases in which the issue is one of activation or precipitation of a dormant disease. It is likewise frequently important for comparative purposes in measuring the visual loss of the injured eye. Any one, whether layman or physician, familiar with the history of cases in compensation procedure will agree that this matter of accurate and complete records cannot be overemphasized.

Sound judgment is not always used in correlating a traumatic lesion of the eye with its claimed results. If the alleged visual disability of a workman with an incentive for deceiving the examiner is not accounted for by ocular changes, as revealed by an adequate examination, the estimate of the amount of disability should be based on what is reasonably in accord with the physical findings. For instance, a small superficial scar of the cornea, which in the light of one's experience cannot, because of its size and location, cause more than 5 or 10 per cent impairment of vision, should not be rated as producing a 50 per cent loss simply because vision is apparently reduced to 20/100. Neither should impairment of vision caused by such conditions as a cataract which is clearly senile, lesions of the fundus due to general disease, old opacities of the cornea or amblyopia due to squint or anisometropia, be charged to a recent injury of the eye simply because the claimant insists that the eye was "all right" before the accident occurred. Yet such claims are being made constantly and, if supported by medical testimony sufficient to raise a slight doubt in their favor, are often successful. Too often the medical opinion in these cases is based not on probabilities but on remote (or imaginary) possibilities. The medical expert's fee for giving his opinion, or testimony, in court should not be contingent on the successful outcome of the case for the side which he is representing.

4. Zimmer, V. A.: Workmen's Compensation Problems, Arch. Ophth. **7**:367 (March) 1932.

Reports of ocular injuries should be concise and the terminology as simple as possible. When writing them, one should remember that they are to be read by laymen. Terms like "O. D." and "O. S." are usually puzzling to the reader. It is just as easy to say "right eye" and "left eye." The same applies to "R. V." and "L. V." If it is necessary to use such terms as ptosis, iridodialysis, aphakia, hyperopia or myopia, which are unfamiliar to the laymen, their meaning expressed in simpler words, in parentheses, should follow.

When, as frequently occurs, an insurance company requests an examination of and opinion concerning an eye that was injured months previously, it is usually interested in one or more of three or four items: (1) the nature and evaluation of the disability, (2) any causal relation between the present condition of the eye and the accident of a given date, (3) indications for further treatment and (4) the prognosis.

Evaluation of the disability is often complicated by the presence of a preexisting or noncompensable defect. It is then often difficult, or impossible, to estimate what percentage of the disability or visual loss is the result of the recent accident. If the patient is seen soon after the accident, old defects are usually recognized as such and noted, but when he is referred months later, the difficulties may be great. The picture is commonly confused by such preexisting conditions as chronic conjunctivitis, scars on the cornea, old adhesions of the iris, opacities on the lens, glaucoma and presbyopia. Often it is vigorously claimed by the examinee that the eye was all right before the accident. The workman is usually given the benefit of any doubt that cannot be resolved.

One occasionally has a case that involves the compensation law doctrine of acceleration or aggravation of a preexisting disease. I refer particularly to those instances of apparently trivial injury, such as a foreign body in the cornea or a slight abrasion, which is followed by gross inflammatory changes in the eye, out of proportion to the severity of the trauma. The uveal tract is most often affected, less frequently the cornea. The more common manifestations are iritis, iridocyclitis, acute choroiditis and persistent ulceration of the cornea, and occasionally there is interstitial keratitis. I believe that the most usual underlying cause is focal infection. Sometimes the source of the infection is in the tonsils or the prostate, but more often, in my experience, it is about the roots of the teeth. When the focus is found and removed, the improvement in the ocular condition is often strikingly prompt and rapid.

Other causes are syphilis and tuberculosis. A few years ago, in discussing a paper on post-traumatic ocular tuberculosis at a meeting of the American Academy of Ophthalmology and Oto-Laryngology, Edward Jackson said: "We have only learned within relatively few years the effects of trauma in producing outbreaks of interstitial keratitis." He expressed the certainty that he had seen several cases of this condition resulting from slight injury of the cornea. We have had 2 or 3 such cases. In 1 case a man aged 23 had had a foreign body removed from the cornea of the right eye three days previously. The eye was slightly red, and there was a faint, deep haze in the lower half of the cornea. This process progressed rapidly. Four days later there was typical interstitial keratitis, with the usual uveitis. Two weeks after the date of the injury the left eye became similarly affected. The man had Hutchinson teeth and a 4 plus Wassermann reaction of the blood. When claim is made for compensation in such cases, the courts have held the employer responsible for the treatment and disability of both eyes.

With regard to the doctrine of aggravation, in the paper to which I have referred, Mr. Zimmer stated:

> Under the theory of compensation principles, firmly fixed by court interpretation in this state, traumatic aggravation or precipitation of a latent condition is the equivalent of causation. Under this theory there are presented to the department a very large number of cases in which one must determine whether and to what extent the injury incited the onroad or development of the diseases which in themselves are capable of reducing or destroying vision. A few of the more important examples include glaucoma, latent tuberculous conditions and syphilis. The latter cases present extremely difficult problems, and in connection with them there arises a great deal of medical controversy.

Sometimes a workman who is sent for treatment of an inflamed eye will say there was a foreign body in the eye a few days before; but, when questioned, he can fix no particular time when the alleged accident occurred and may admit that he thought there was something in the eye because it was uncomfortable. If the case is clearly not compensable, especially if the injury is serious, the facts should be reported to the employer (or the insurance carrier), so that he may have the choice of authorizing or refusing further medical care.

671 Broad Street.

LIPEMIA RETINALIS IN THE NONDIABETIC PATIENT

CECIL W. LEPARD, M.D.
DETROIT

Since certain changes in the appearance of the arteries and the veins of the retina were described by Heyl [1] as lipemia retinalis in 1880, various other authors have reported in detail some 50 cases up to the present time and thus have made lipemia retinalis well known to the ophthalmologist. Records from larger clinics and hospitals indicate that it is more commonly observed than reported in the literature. With the exception of Wagener's case,[2] reported in 1922, the condition has been observed entirely in patients with diabetes. This has led authors of textbooks to give the reader the impression that diabetes is the only disease wherein this phenomenon may be seen in the fundus.

The level of blood lipids at which lipemia retinalis is first recognized and the level at which it disappears have been carefully determined by several authors (Chase,[3] Kollner,[4] Parker and Culler,[5] McKee and Rabinowitch [6] and Lepard [7]). Although there is some variation in these levels as determined by the different authors, lipemia retinalis appears approximately when blood fats increase to above 3.5 per cent and disappears when these fats decrease to below 2.5 per cent.

Hardy [8] stated the belief that the occurrence of lipemia retinalis is of bad prognosis. However, later it was found that patients with this condition have the same expectancy as patients with severe diabetes. In a series of 38 cases, Parker and Culler [5] noted that acidosis was present in all but 2 patients. Marble and Smith [9] stated that lipemia retinalis occurs usually, if not exclusively, in patients with acidosis or in persons who have recently had acidosis.

Lipemia retinalis was observed over a period of several years in an otherwise well person showing a persistent high concentration of fats in the blood. Complete studies on the blood were made and component lipids of plasma and erythrocytes determined. Other clinical and metabolic studies were made by Bernstein, Williams, Hummel, Shepherd and Erickson.[10] From the results of these some of the questionable phases of lipemia retinalis may be studied, and a correlation between the ophthalmoscopic findings and the components of the emulsion of the lipids in the blood may be made. It is well known that a high level of blood lipids does not result in visible changes in the fundi in most cases. While the permeability of the arteries and the veins may be one factor, component parts of the emulsion are probably another. Further investigation may be necessary in order to determine other reasons for emulsification of the lipids of the blood in lipemia. Lipemia retinalis is simply the visual manifestation of an emulsion of blood fats.

REPORT OF A CASE

A 6 year old white boy was admitted to Children's Hospital, Nov. 16, 1936, because of abdominal cramps. There was no history of visual disturbance, ocular pain or inflammation. Routine examination revealed an enlarged spleen and liver and a mild fecal impaction, which was later relieved by a cleansing enema. Samples of blood taken for Wassermann and agglutination tests had the appearance of a creamy emulsion. Wassermann, Kahn and Mantoux reactions were negative. Roentgenograms of the abdomen showed a slightly enlarged liver and a considerably enlarged spleen. The result of a galactose tolerance test of hepatic function

Dextrose Tolerance Test, Mg. per 100 Cc.

Fasting	91
½ hour	250
1 hour	175
2 hours	103
3 hours	90

was normal. Roentgenograms of the long bones showed no changes. Repeated examinations of the urine failed to reveal sugar, acetone, diacetic acid and albumin. The blood cell count was normal, and the basal metabolic rate was within normal limits.

Vision in both eyes was 6/6. Examination of the fundi showed severe lipemia retinalis in each eye. With a slit lamp the anterior segment of each eye appeared

Candidate's thesis for the American Ophthalmological Society, 1943.

1. Heyl, A. G.: Philadelphia M. Times **10**:318, 1880; Tr. Am. Ophth. Soc. **3**:54, 1880.
2. Wagener, H. P.: Am. J. Ophth. **5**:521, 1922.
3. Chase, L. A.: Diabetic Lipaemia Retinalis: Report of Case, J. A. M. A. **97**:171 (July 18) 1931.
4. Kollner, K.: Centralbl. f. prakt. Augenh. **34**:212, 1910; Ztschr. f. Augenh. **27**:411, 1912.
5. Parker, W. R., and Culler, A. C.: Am. J. Ophth. **13**:573, 1930.
6. McKee, S. H., and Rabinowitch, I. M.: Canad. M. A. J. **15**:530, 1931.
7. Lepard, C. W.: Am. J. Ophth. **16**:12, 1933.
8. Hardy, W. F.: Tr. Am. Ophth. Soc. **19**:229, 1921; Am. J. Ophth. **7**:39, 1924.
9. Marble, A., and Smith, R. M.: Blood Lipids in Lipaemia Retinalis, Arch. Ophth. **15**:86 (Jan.) 1936.
10. Bernstein, S. S.; Williams, H. H.; Hummel, F. C.; Shepherd, M. L., and Erickson, B. H.: J. Pediat. **14**: 570, 1939.

normal. The blood stream of the smaller vessels of the conjunctiva had the appearance of a creamy emulsion, which made the identification of blood cells difficult.

The patient was placed on a measured low fat diet consisting of 45 Gm. of protein, 18 Gm. of fat and 210 Gm. of carbohydrate. The high fat diet consisted of 45 Gm. of protein, 100 Gm. of fat and 30 Gm. of carbohydrate. Results are shown in table 1.

TABLE 1.—*Analyses of the Blood Lipids in a Case of Essential Hyperlipemia* *

	Low Fat Diet	High Fat Diet
Plasma (mg. per 100 Cc.)		
Total lipid	3,159	9,370
Phospholipid	436	833
Neutral fat	2,166	7,636
Free cholesterol	129	199
Cholesterol esters	428	702
Erythrocytes (mg. per 100 Gm.)		
Total lipid	647	930
Phospholipid	481	383
Neutral fat	51	447
Free cholesterol	95	80
Cholesterol esters	20	15

* From the Children's Hospital, Children's Fund of Michigan Research Division.

He was maintained on a low fat diet, under which the spleen and the liver gradually decreased in size. This boy continued in school with normal activities and on Feb. 12, 1943 returned to the hospital, at which time a sample of blood was taken. The results of these examinations closely parallel those of the original studies, as seen in table 2.

TABLE 2.—*Low Fat Diet Consumed at Home*

Plasma (mg. per 100 Cc.)	
Phospholipid	487
Free cholesterol	162
Cholesterol esters	351
Erythrocytes (mg. per 100 Gm.)	
Phospholipid	388
Free cholesterol	89
Cholesterol esters	95

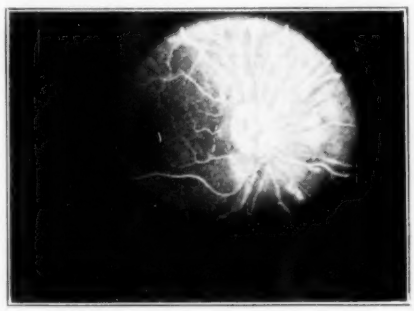
Lipemia retinalis was present during the entire time the patient was under observation. On a low fat diet it could be recognized only in the smaller divisions of the central artery and vein in the more peripheral portions of the fundus of each eye. With a high fat diet the arteries and the veins throughout their entire extent were increased to twice their size and approached a common creamy color, so as to be indistinguishable from each other.

TABLE 3.—*Percentage of Total Fat*

	Low Fat Diet		High Fat Diet
Plasma			
Neutral fat	68	increased	81
Phospholipid	14	decreased	9
Free cholesterol	4	decreased	2
Cholesterol esters	14	decreased	7
Erythrocytes			
Neutral fat	8	increased	48
Phospholipid	74	decreased	41
Free cholesterol	14	decreased	9
Cholesterol esters	3	decreased	2

From table 1 it is seen that there was a more striking increase in the total lipid content of the plasma than of the erythrocytes. The percentage of neutral fat in both the plasma and the erythrocytes (table 3) showed a distinct increase when the patient was on the high fat diet, while at the same time the percentages of cholesterol esters, phospholipid and free cholesterol were slightly decreased. The percentage of neutral fat of the erythrocytes was increased far more than that of the plasma when fat was added to the diet. When the emulsion contained a relatively high percentage of neutral fat, the lipemia retinalis was severe. When the state of the emulsion changed under a low fat diet containing a relatively small percentage of neutral fat, the ophthalmoscopic examination showed only a slight degree of lipemia retinalis.

The patient had acidosis for a small part of the time while under observation. Acidosis may have resulted from abnormal fat metabolism, however, in this and in my previous case.[7]



A photograph showing the appearance of the fundus of the patient on a high fat diet. Note the increase in size of the blood vessels and how they stand out in relief beyond the disk. This photograph was supplied by Dr. F. L. Ryerson.

It had no relation to the occurrence or the degree of lipemia retinalis. Acidosis seems to be of secondary interest.

Lipemia retinalis has been observed in a nondiabetic person with lipemia, whose renal and hepatic functions were normal (Holt, Aylward and Timbus [11]). For this patient also neutral fat levels were high. The authors stated the belief that neutral fat was not normally disposed of and was accumulated in the phagocytic cells of the reticuloendothelial system, which resulted in an enlargement of the liver and the spleen.

SUMMARY

Although lipemia retinalis is usually seen in the young person with severe diabetes, it is a phenomenon resulting from a rise in the level of the blood lipids and may occur in the presence of normal carbohydrate metabolism.

Blood lipids composed of a high percentage of neutral fat seem more likely to produce characteristic retinal changes than do those composed of a low percentage of neutral fat.

11. Holt, L. E.; Aylward, F. X., and Timbus, H. G.: J. Clin. Investigation **15**:451, 1936.

JUVENILE AMAUROTIC FAMILIAL IDIOCY

ITS OCULAR PATHOLOGY

ISADORE GIVNER, M.D., AND LEON ROIZIN, M.D.
NEW YORK

Cerebromacular degeneration, or the juvenile form of amaurotic familial idiocy, was first described by Batten [1] in 1903. The clinical features of this disease were progressive loss of the intellectual faculties, loss of vision and loss of motor power. This condition differed from Tay-Sachs disease (infantile amaurotic familial idiocy) by the fact that cases did not occur predominantly in the Jewish race, that blindness was not associated with the same typical picture of the fundus, that its first clinical manifestations appeared at a later age and that the progress was slower. In 1905 Vogt [2] described the morbid anatomy, and Spielmeyer,[3] in the same year, reported cases of a condition resembling retinitis pigmentosa in which the pigmentary changes were thought at first to be a complication of the disease.

In 1908 Stock [4] first published the histologic changes in the eye in 3 of Spielmeyer's original cases. He noted that the optic nerves were normal and expressed the belief that this is a characteristic which distinguishes it from Tay-Sachs disease and retinitis pigmentosa. The only case in this country in which the histologic changes in the eye were described is that reported by Wandless,[5] but no photographs were included.

Through permission of Dr. Rayford K. Adams, an unusual opportunity was had to observe 2 patients, a brother and a sister, with this condition. The girl died Sept. 15, 1942, and on her death the globes and brain were secured for study. At about this time a second patient died of the disease, and material was likewise made available for histologic study.

Since we studied the fundi and made photographs only of the girl, the usual histopathologic studies made in this case are reported in case 1. Material in the second case was kept for investigation of the histochemical nature of the lipids.

REPORT OF CASES

CASE 1.—D. N., a girl, was admitted to the New Jersey State Village for Epileptics on Aug. 29, 1940. She was the sixth of 7 children. The younger brother of the patient also has cerebromacular degeneration and is still a patient in the Village. Her history, taken from the records of the institution, is as follows: "The patient was born May 31, 1923. She was normal until the age of 4 years, when she began to have trouble with vision. She was thought to be mentally deficient at that time. She had 'spasms' shortly after birth but had no more convulsions until the age of 13 years. Her eyesight faded entirely. She attended school for the blind for one and a half years and learned to read Braille."

Ophthalmoscopic Examination.—The lens of the right eye had fine gray opacities at the posterior pole; the lens of the left eye was normal. Convergent strabismus with rotary nystagmus was present in the left eye. Both fundi showed optic nerve atrophy. The vessels were very thin. The maculas had areas of depigmentation and condensation. Scattered over the fundi of both eyes were clumps of pigment. Some resembled bone corpuscles in shape; others were less well defined (fig. 1). Vision was limited to light perception. The patient died Sept. 15, 1942, at the age of 19 years.

Histopathologic Study of Eyes.—The globes were fixed in a 10 per cent concentration of dilute solution of formaldehyde for forty-eight hours and then in Zenker's solution. They then were dehydrated in the usual way and fixed in both pyroxylin and paraffin. The entire retina was involved, but to different degrees. No uniformity in the intensity of involvement was noted.

Read at a meeting of the New York Academy of Medicine, Section of Ophthalmology, Nov. 15, 1943.

From the Department of Neuropathology of the New York State Psychiatric Institute and Hospital.

1. Batten, F. E.: Cerebral Degeneration with Symmetrical Changes in the Maculae in Two Members of a Family, Tr. Ophth. Soc. U. Kingdom **23**:386, 1902-1903.

2. Vogt, H.: Ueber familiäre amaurotische Idiotie und verwandte Krankheitsbilder, Monatschr. f. Psychiat. u. Neurol. **18**:161 and 310, 1905.

3. Spielmeyer, W.: Ueber familiäre amaurotische Idiotien, Neurol. Centralbl. **24**:620, 1905; Weitere mitteilung über eine besondere Form von familiärer amaurotischer Idiotie, ibid. **24**:1131, 1905; Ueber familiäre amourotische Idiotien, Arch. f. Psychiat. **40**: 1038, 1905; Klinische und anatomische Untersuchungen über eine besondere Form von familiärer amaurotischer Idiotie, in Nissl, F., and Alzheimer, A.: Histologie und Histopathologie, Jena, G. Fischer, 1908, vol. 2, p. 193.

4. Stock, W.: Ueber eine bis jetzt noch nicht beschriebene Form der familiär auftretenden Netzhautdegeneration bei gleichzeitiger Verblödung und über typische Pigmentdegeneration der Netzhaut, Klin. Monatsbl. f. Augenh. **46**:225, 1908.

5. Wandless, H. W.: Amaurotic Family Idiocy: A Preliminary Report of Three Cases, New York M. J. **84**:953, 1909.

Here and there small portions were less severely damaged than the retina around them.

The most prominent change was the diminution in width of the internuclear layer; in some portions the inner and the outer nuclear layer were juxtaposed (fig. 2). The choroid appeared normal (fig. 3). The pigment epithelium showed many changes. An irregular loss of pigment within the cells was associated with scattering of its granules at the site of the rods and cones in large, dense spherical masses. The pigment cells at the same time became flattened, less regularly arranged and in some places heaped into two or more layers. The nuclei of the pigment epithelium were fairly normal in appearance but in places seemed shrunken and irregular and stained deeply. The most striking change was in the depigmentation. The rods and cones seemed to lie in an organized mass of detritus, the individual elements being practically nonexistent, except here and there. The pigment and nuclei, like those of the pigment epithelium, were scattered through this mass of tissue. This change did not occur post mortem, as the globes were fixed soon after death (fig. 4). In many places the outer limiting membrane could not be recognized. The outer nuclear layer was notably affected, as evidenced by irregularity of arrangement; smaller, shrunken cells, and sparseness. The inner nuclear layer was less affected but was also involved, and, as was said before, the internuclear layer was frequently absent (fig. 5). The ganglion cell layer showed no diminution in number of cells, but structural changes were observed. Some ganglion cells were swollen and others spherical; some

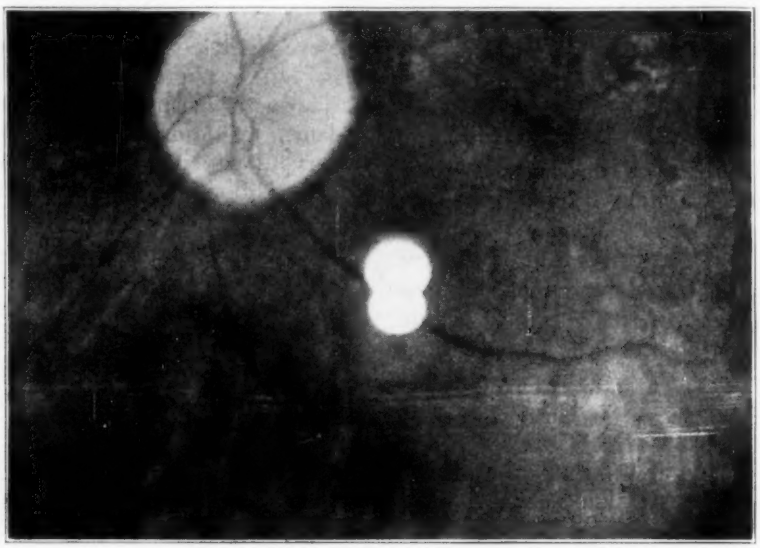


Fig. 1.—Photograph of the fundus in case 1, showing optic nerve atrophy, narrowed vessels and clumping of pigment.

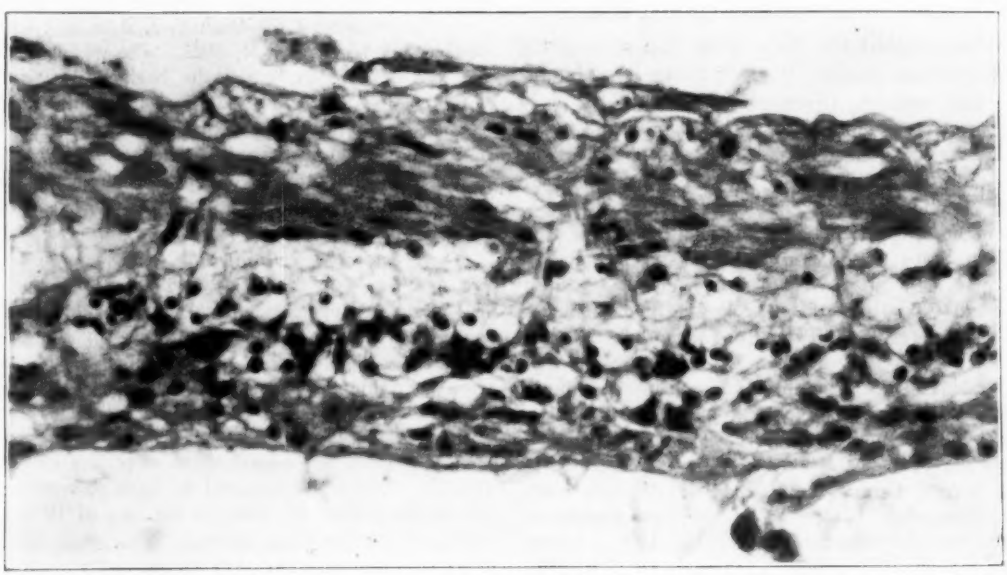


Fig. 2.—Juxtaposition of the inner and the outer nuclear layers, with no remaining internuclear layer.

Fig. 3.—Normal choroid.

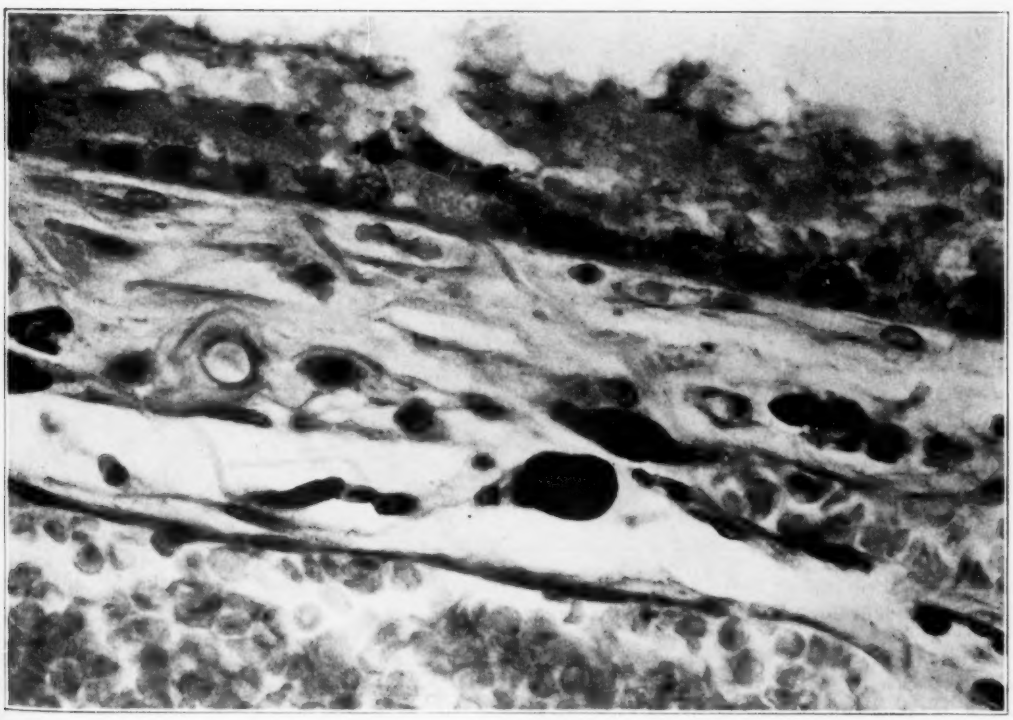


Fig. 4.—Scattering of pigment of the pigment epithelium and replacement of the layer of rods and cones by an organized mass of detritus.

contained granular masses; others, eccentric nuclei, and in some, vacuoles, as described in cases of Mayou and Ichikawa, were noted (fig. 6). The nerve fiber layer showed an increase of glial tissue, as did most of the retina proper, where the neuroglia replaced degenerated layers. The retinal vessels showed questionable splitting of the elastic fibers. Stock noted slight endarteritis in Spielmeyer's cases.

The optic nerve showed definite areas of atrophy, as shown by the Smith-Quigley stain (fig. 7).

Torrance described 3 cases, in all of which optic nerve atrophy was present. Optic nerve atrophy was absent in Spielmeyer's cases. Brooks, in the case published by Wandless, observed no ganglion cells in the retina or nerve fibers in the optic nerves, but Marchi's method revealed droplets of fat in the optic nerve, which he concluded indicated degenerated fibers.

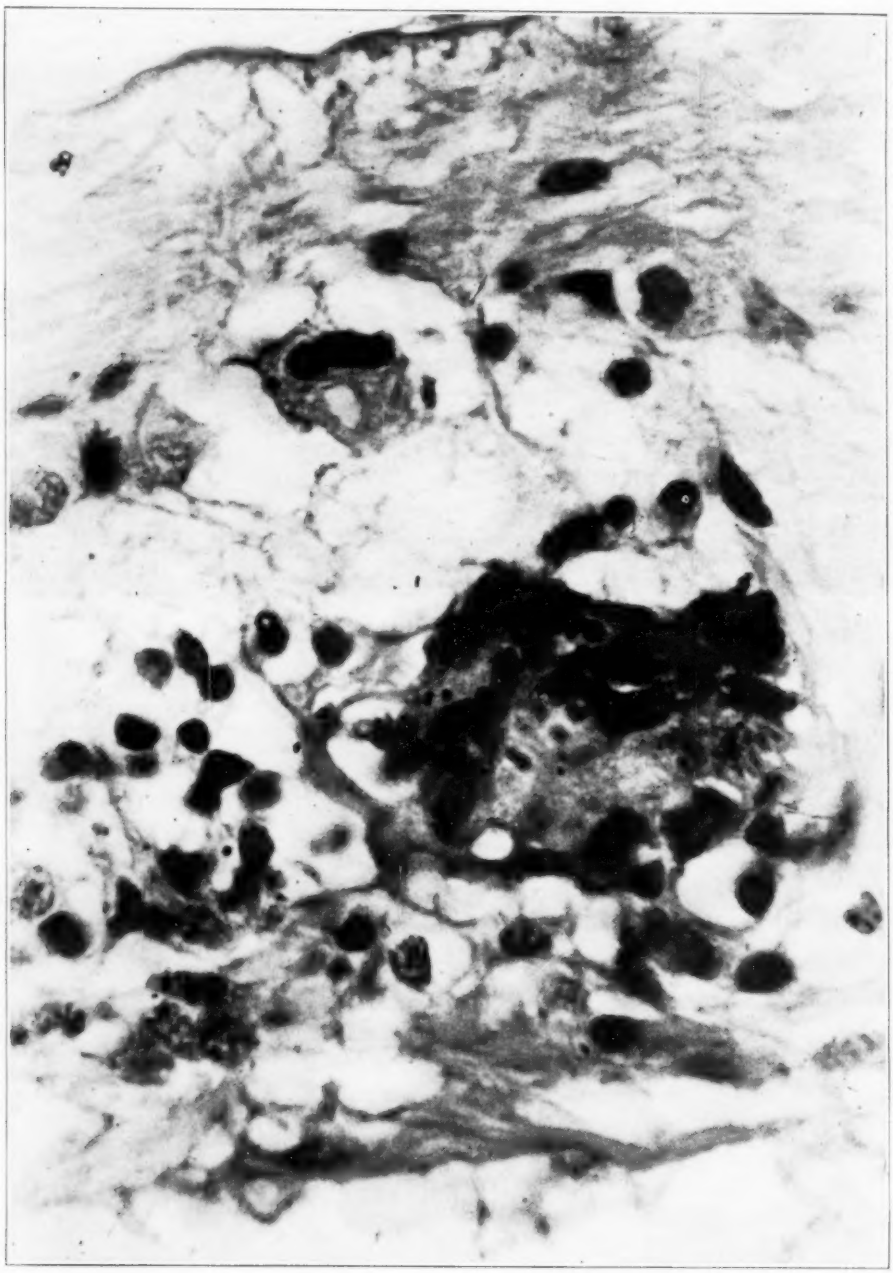


Fig. 5.—Typical section of the retina, showing disorganization with resulting loss of the internuclear layer and clumping of pigment.

Examination of a ganglion cell accompanying a ciliary nerve showed no pathologic changes.

Various observers have attempted to determine which change was primary and which secondary and have tried to draw conclusions

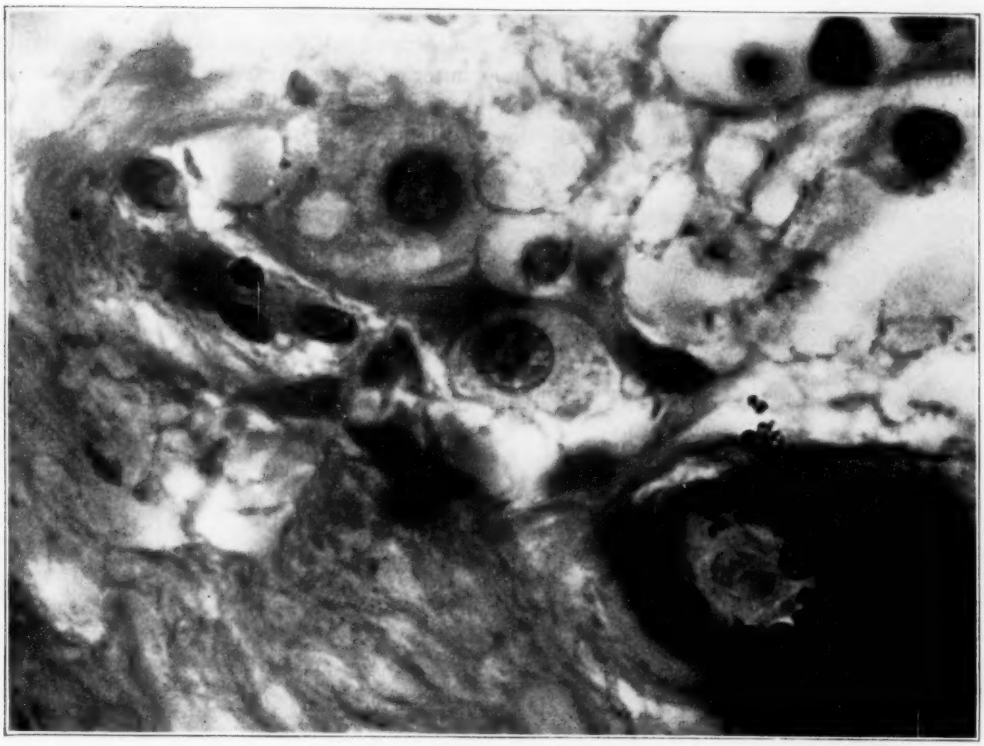


Fig. 6.—Ganglion cells of the retina, one of which shows two large vacuoles in the cytoplasm.

Fig. 7.—Optic nerve atrophy, as demonstrated by the Smith-Quigley stain.

from their own cases. Stock concluded that the changes were due to primary degeneration of the neuroepithelium and secondary proliferation, combined with regressive changes in the pigment epithelium. Ichikawa [6] interpreted the changes as a uniform degeneration of all the nervous elements of the retina—similar to that which occurs with retinitis pigmentosa. Holmes and Paton [7] expressed the belief that the earliest lesion was a breaking up of the rods and cones, associated with rarefaction and narrowing of the outer nuclear layer, the cell bodies to the outer nuclear layer being affected first and the afferent and efferent processes next. The degenerated external layers of the retina were replaced by proliferating neuroglia, followed by overgrowth of the pigment epithelium, with invasion of the degenerating retinal layers. The changes in the pigment cell layer were said to be secondary to the lesion in the other layers of the retina.

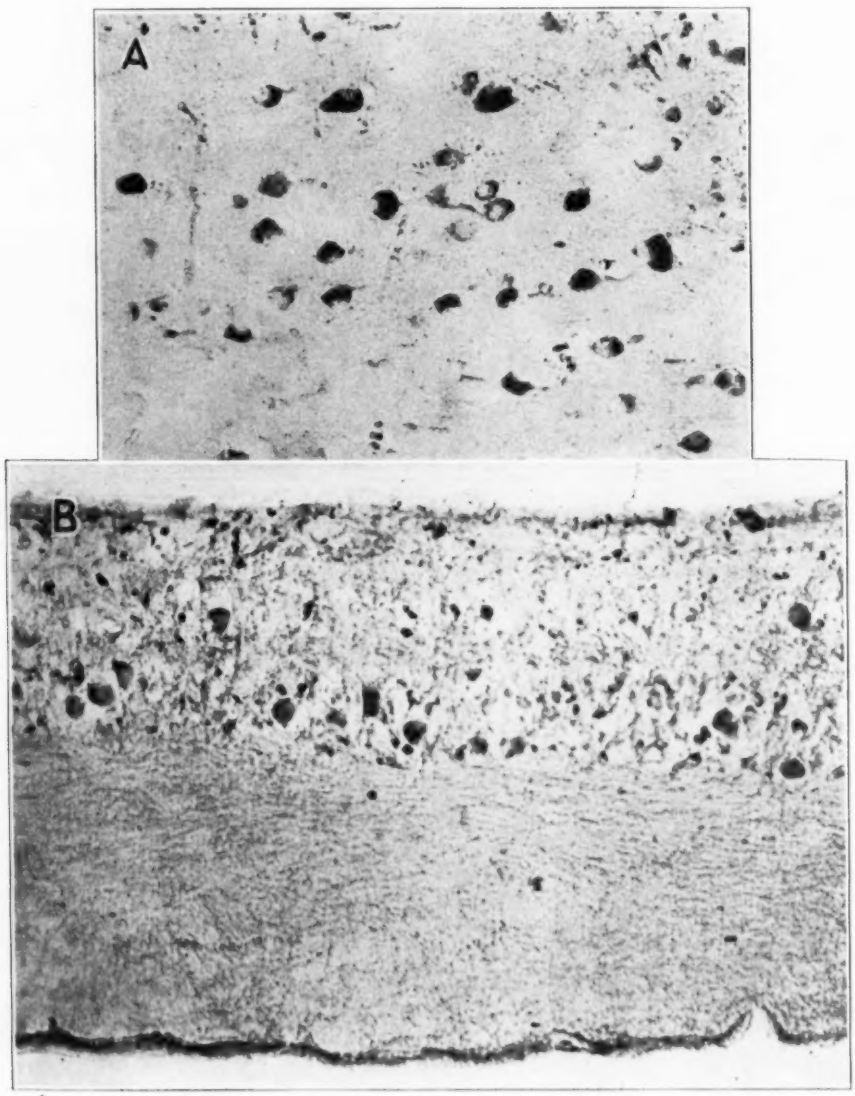


Fig. 8.—Intracellular fatty products (*A*) in the ganglion cells of the cerebral cortex and (*B*) in the ganglion cells of the retina. Sudan black B; low power magnification.

6. Ichikawa, K.: Ueber eine der amaurotischen familiären Idiotie verwandte Krankheit mit histologischer Beschreibung, nebst einem Beitrag zur Kenntnis der Beziehung zwischen erblichen Augenkrankheiten und zur Pathogenese der primären Cyste der Pars ciliaris, Klin. Monatsbl. f. Augenh. **47**:73, 1909.

7. Holmes, G., and Paton, L.: Cerebro-Macular Degeneration (the Juvenile Form of Amaurotic Family Idiocy), Tr. Ophth. Soc. U. Kingdom **45**:447, 1925.

Mayou,[8] who examined the eye in 1 of Batten's cases, in which there was complete disappearance of the ganglion cells, expressed the belief that the disease commenced here, where the cells swelled, lost their Nissl substance, became vacuolated and eventually disappeared. This was followed by edema of the internuclear layer, by gradual disappearance of the outer nuclear layer and of the rods and cones and, finally, by pigmentation. Greenfield and Holmes[9] best summarized the seeming discrepancy by concluding that all the cases reported can be placed in one of three groups:

(1) Cases similar to those of Stock, Bielschowsky and Holmes, in which only the outer layers of the retina suffer and changes in the inner layers are probably secondary; (2) cases of Mayou, Didé and Torrance, with changes in the inner layers, especially the ganglion cells, sometimes associated with changes in the rods and cones, and (3) cases of Brooks and Ichi-

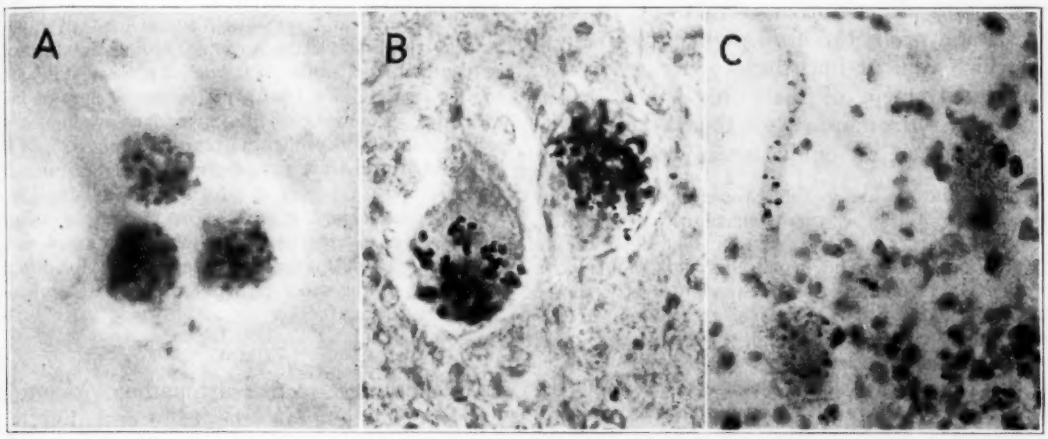


Fig. 9.—Prelipid products (*A*) in the ganglion cells of the cerebral cortex, (*B*) in the Purkinje cells of the cerebellum (modified Schaffer method) and (*C*) safranophilic granules in a Purkinje cell and its apical dendrite (safranin stain). High power magnification.

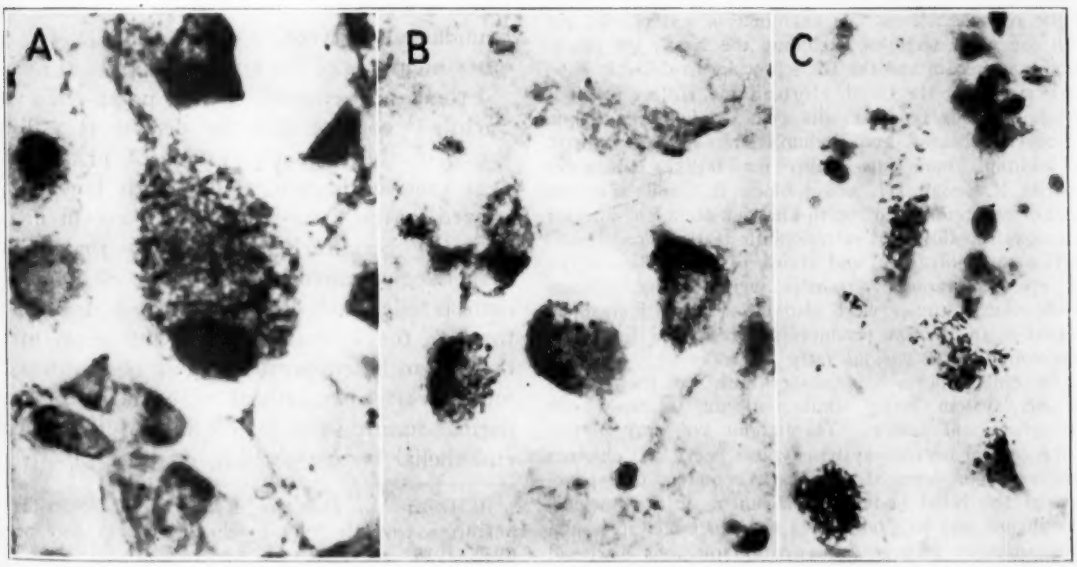


Fig. 10.—Ganglion cells of the retina (*A*) argentophilic products (modified Bielschowsky method), (*B*) prelipid products (modified Schaffer method) and (*C*) safranophilic granules mixed with some pigment (safranin stain). High power magnification.

8. Mayou, M. S.: Cerebral Degeneration, with Symmetrical Changes in the Maculae, in Three Members of a Family, Tr. Ophth. Soc. U. Kingdom **24**:142, 1905. Batten, F. E., and Mayou, M. S.: Family Cerebral Degeneration with Macular Changes, Proc. Roy. Soc. Med. (Ophth. Sect.) **8**:70, 1914-1915.

9. Greenfield, J. G., and Holmes, G.: The Histology of Juvenile Amaurotic Idiocy, Brain **48**:183, 1925.

kawa, in which all layers are severely degenerated.

Greenfield and Holmes concluded that the retinal changes may be defined as a primary change in the ganglion cells combined with independent degeneration of the outer neurotic elements of the retina, which in some cases extends to the inner nuclear and inner reticular layers. This degeneration is followed by glial sclerosis and proliferation of pigment epithelium cells, which wander into the degenerated tissue.

The involvement of the outer layers distinguishes the retinal lesions of the juvenile from the infantile type of familial amaurotic idiocy.

CASE 2.—The disease in this case was similar to the Batten-Mayou type of cerebromacular degeneration. The patient, a first child, was born on Sept. 30, 1928. In 1935, at the age of 7, he began to have trouble with his eyes. The macular region of the right eye presented a picture of degeneration. Physical examination at this time revealed nothing abnormal. At the age of 8 years convulsions appeared.

He was admitted to the Village on April 8, 1942, at the age of 14 years. Convergent strabismus with nystagmus was noted. Vision was limited to light perception. He was disoriented for time, place and persons. He had frequent convulsions and died on Sept. 20, 1942. Simultaneous comparative histopathologic and histochemical studies were made on the ganglion cells of the central nervous system and those of the retina. Microscopic examination was carried out with the usual technics, including the Nissl, the hematoxylin and eosin and the Bielschowsky modified method for nerve cells; the Cajal, Hortega and Holzer methods for the various types of glia cells; Spielmeyer's stain for myelin sheaths, and Bodian's stain for neurofibrils. In addition, numerous stains for fatlike substances (scarlet R, sudan III, sudan black B, osmic acid and the Lorrain-Smith and Smith-Dietrich technics) and for hematoxylinophilic and safranophilic bodies (iron hematoxylin and safranin) and stains of the aniline series for special granular products were applied. Some histochemical studies were also based on histochemical properties of the fat products for polarized light and their solubility in various fatty solvents.

The chief microscopic observations on the central nervous system being similar in the 2 cases, one description will suffice. Throughout the gray matter of the central nervous system typical nerve cell changes were seen—pronounced tigrolysis, complete disappearance of the Nissl bodies, displacement of the nucleus, pear-shaped and balloon-shaped neurons, satellitosis and neuronophagia. Silver impregnation methods disclosed various stages of fibrillary changes in the neurons and their processes. Involvement of nerve cells in the cortex and in the various gray centers of the brain varied in intensity and extent. The cerebellum disclosed slight degeneration of the elements of the granular layer, being more pronounced in the Purkinje cells.

The cytoplasmic network of altered cells was filled at times with a granular material, which was particularly well demonstrated in frozen sections stained by methods for fatlike substances; these granules stain orange or mat red with scarlet R and sudan III, at times slightly greenish with sudan black B, grayish with osmic acid and faintly bluish with nile blue sulfate. Lipid granules were present, also, in the apical processes of the neurons of the cortex; they were particularly conspicuous in the Purkinje cells, which showed the most advanced stages of degeneration.

The solubility of the fatlike products were tested with various solvents. This material failed to dissolve after several hours (two to six hours) of immersion in cold alcohol, ether and acetone respectively. After treatment with xylene and benzene for one-half to one hour, the granules stained much more faintly with scarlet R, sudan III and sudan black B. Hot alcohol, a mixture of chloroform and methanol, as well as pyridine, appeared to dissolve the granules completely. Sections stained with safranin [10] as well as with stains of the aniline series [11] and iron hematoxylin,[12] showed in some cells the presence of fine granular products (safranophilic or anilinophilic and prelipid granules). Figures 8, 9 and 10 illustrate the presence of intracellular fatlike material and granular products in the ganglion cells of the cortex and of the retina. The histochemical reactions, as evidenced by the aforementioned methods, seemed to point to the presence in both the brain and the retina of the same type of lipids, i. e., a mixture of phosphatides and cerebrosides.

COMMENT

With respect to the histopathologic and clinical interrelations or the differentiation of the several types of amaurotic familial idiocy, we agree with Globus [13] that "while minute studies in certain cases may often disclose . . . deviations from the typical pathology of clinical pictures, they nonetheless fail to establish sharp boundaries between a given case . . . and other members of the entire group."

From an etiopathogenic point of view, Sachs's [14] concept that the disease is a disturbance of the nervous system of primary and degenerative character still stands true even if viewed in the light furnished by recent investigations. Schaffer's [15] hypothesis that it is a heredodegenerative disease restricted only to neuroectodermal derivatives and transmitted through the hyaloplasm does not seem in our opinion to be supported by our observations, in view of the involvement of elements of mesodermal nature—the microglia and the reticuloendothelial system—which has been reported by

10. Didé, M., and van Bogaert, L.: Sur l'idiotie amaurotic juvénile (type Spielmeyer-Vogt), Rev. neurol. **69**:1, 1938.

11. Jervis, G. A.; Roizin, L., and English, W. H.: Juvenile Amaurotic Idiocy, Psychiatric Quart. **16**:132, 1942.

12. Schaffer, K.: Tatsächliches und Hypothetisches aus der Histopathologie der infantile-amaurotischen Idiotie, Arch. f. Psychiat. **54**:570, 1921-1922.

13. Globus, J. H.: Amaurotic Family Idiocy, J. Mt. Sinai Hosp. **9**:451, 1942.

14. Sachs, B.: On Amaurotic Family Idiocy: A Disease Chiefly of the Gray Matter of the Central Nervous System, J. Nerv. & Ment. Dis. **30**:1, 1903.

15. Schaffer, C.: General Significance of the Tay-Sachs Disease, Arch. Neurol. & Psychiat. **14**:731 (Dec.) 1925.

other investigators (Pick and Bielschowsky[16]; Bielschowsky[17]; Jervis, Roizin and English,[11] and others). This hypothesis, however, remains open for further study (Sachs[18]; Hassin[19]; Globus,[20] and others). Gordon's,[21] and particularly Marburg's,[22] assumption that an endogenous factor, of hormonal origin (adrenal, medullary portion, and thymus), is involved and that the "lack of respective esterases is the probable cause of the damage of the nervous system" needs confirmation.

In conclusion, the histochemical reactions as evidenced by our investigation, in both the retina and the brain, point to the presence in the involved cells of the same type of lipids, i. e., a mixture of phosphatides and cerebrosides.

108 East Sixty-Sixth Street.

722 West One Hundred and Sixty-Eighth Street.

16. Pick, L., and Bielschowsky, M.: Ueber die lipoidzellige Splenomegalie (Typus Niemann-Pick) und amaurotische Idiotie, Klin. Wchnschr. **6**:1631, 1927.

17. Bielschowsky, M.: Amaurotische Idiotie und lipoidzellige Splenohepatomegalie, J. f. Psychol. u. Neurol. **36**:103, 1928.

18. Sachs, B.: Amaurotic Family Idiocy and General Lipoid Degeneration, Arch. Neurol. & Psychiat. **21**: 247 (Feb.) 1929.

19. Hassin, G. B.: Amaurotic Family Idiocy (Infantile Type of Tay-Sachs), in Histopathology of the Peripheral and Central Nervous Systems, ed. 2, New York, Paul B. Hoeber, Inc., 1940, p. 353; Histopathologic Observations on the Changes of the Eyes in a Case of Amaurotic Family Idiocy (Infantile Type of Tay-Sachs), J. Mt. Sinai Hosp. **9**:536, 1942; Niemann-Pick Disease: Pathologic Study of a Case, Arch. Neurol. & Psychiat. **24**:61 (July) 1930.

20. Globus, J. H.: Amaurotic Family Idiocy, in Penfield, W.: Cystology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 3, p. 1166; footnote 13.

21. Gordon, A.: Cases Allied to Amaurotic Family Idiocy, with Remarks on the Genesis of the Affection, New York M. J. **85**:294, 1907.

22. Marburg, O.: Studies on the Pathology and Pathogenesis of Amaurotic Family Idiocy, Am. J. Ment. Deficiency **46**:312, 1942.

INTRACAPSULAR EXTRACTION OF SENILE CATARACT

MATHRA DAS PAHWA, M.D.
LAHORE, INDIA

I have performed 170,000 operations for cataract, almost all of them intracapsular extractions, and the method which I have followed throughout was devised by me shortly after I began ophthalmic surgery.

The controversy as to the use of the intracapsular or the extracapsular operation is always going on in all countries; Indian surgeons as a whole, especially the ophthalmic surgeons in the Punjab, have always favored the intracapsular method, while in some localities the extracapsular operation is the rule, although some surgeons in Europe have adopted the intracapsular operation as a routine in their clinics.

The advantages of the intracapsular method may be presented as follows: (1) It does not require two operations; (2) the operation can be performed even when the cataract is not completely mature; (3) as there are no shreds of capsule or lens matter to be caught in the edges of the wound, the chances of subsequent iritis and cyclitis are reduced to the minimum, and (4) ultimate vision after an intracapsular operation is much better than after extracapsular extraction.

There is one disadvantage, loss of vitreous, which, if it does occur, is not a serious matter. It is this accident, however, which has led many surgeons to abandon the operation.

I have perfected intracapsular delivery of the lens by the tumbling method, and all the ophthalmic surgeons who have visited my clinic are impressed with the rarity of the loss of vitreous.

OPERATION

This operation is the method of choice with me and many surgeons have been trained in my clinic because of its remarkable simplicity, its freedom from complications and little instrumentation. It is suitable for all types of senile cataract, whether mature or not, hard or morgagnian.

As this operation is simple, it requires few instruments. These consist of speculum, a pair of fixation forceps, cataract knife, iris forceps, a pair of iris scissors, two squint hooks, iris spatula, scoop and cystotome. The last three instruments are kept simply as a precautionary measure.

An assistant who knows what is expected of him at each stage is necessary.

Read before the Indian Medical Association, Srinagar, Kashmir, India.

Anesthesia.—A 2 per cent solution of butacaine sulfate, 1 drop at a time, is instilled three or four times at three minute intervals before the operation. It gives a fairly complete anesthesia. I do not use facial block.

Section.—The patient is requested to lie down on the table. After insertion of the speculum, the eyelashes of the upper lid are cut with a pair of scissors, and the eye is irrigated with a 1:5,000 solution of mercury bichloride. The eye is fixed with fixation forceps. The knife is inserted at 9 o'clock (right eye) in the cornea at the side of the limbus, and is carried straight across the anterior chamber and brought out exactly at 3 o'clock. With the left eye, the section is from 3 to 9 o'clock. With gentle sawing movements, the knife is carried straight upward, and the section is finished about 1 mm. below the upper border of the cornea.

It will be obvious from this description that I believe in a low section; the scar is hidden by the lid and therefore is not a disadvantage. Also if a low section is made the delivery of the lens is much easier.

With beginners, it is a common fault to press the eyeball with the fixation forceps; this should always be guarded against, and just before the section is finished the fixation forceps should be removed.

Iridectomy.—I perform iridectomy in all cases; this, again, makes the delivery of the lens easier. The iris is pulled out through the corneal wound with the iris forceps and cut with iris scissors.

Delivery of Lens.—At this stage the speculum is removed, and the assistant lifts the upper eyelid with a strabismus hook held in his right hand and pulls the lower lid down with the fingers of his left hand. The surgeon places his left thumb just above the eyebrow and slightly pulls it up, and in the other hand he holds the other strabismus hook. This hook is now applied to the sclera 2 mm. below the junction, at about 6 o'clock. It is slowly drawn up toward the limbus with very slight backward pressure, which movement dislocates the lens. It is a mistake to apply too much pressure; a gentle touch is enough. If with the first attempt the lens is not dislocated, the pressure should be repeated. As soon as the lens is dislocated, the lower border of the lens will be seen in the anterior chamber. With the hook the lower pole of the lens is lifted up still farther until it presents in the corneal section.

It will be gathered from the foregoing description that the lens turns a complete somersault before it is delivered; that is, the lower pole of the lens comes out first through the corneal wound; this constitutes "tumbling of the lens." I was the first to attempt extraction of the ordinary hard senile cataract by this technic. I have trained many operators from all parts of the world who follow this method, and I have

no doubt that the operation will gain still more in popularity.

After the lens is delivered, the iris is carefully freed from the edges of the corneal wound and replaced either with the strabismus hook or with an iris spatula. The upper lid is lowered gently; the patient is warned in advance not to close his eyes tightly or in any way squeeze his eye. The eye is covered with a piece of cotton wool, and both eyes are bandaged.

After-Treatment.—The patient should lie straight on his back and remain quite still for the first four to six hours, after which he is permitted to turn toward the normal eye. Unless there are indications which demand examination of the eye earlier, I generally inspect the eye on the seventh day, when the wound is healed, and the fellow eye is left uncovered.

On the tenth day, in the absence of complications, such as prolapse of the iris, ununited corneal wound or sepsis, the bandage is removed from the eye which has been operated on and the eye covered with a green shade.

SUMMARY

Like any other surgical procedure, a little patience and considerable practice are required before the art of neat delivery of the lens is mastered. Perseverance is essential, but if the technic is once mastered, the surgeon will have a simple, efficient and safe method of doing his cataract operations.

5 Temple Road.

INTRAOCULAR PRESSURE AND ITS RELATION TO RETINAL EXTRAVASATION

JOSEPH IGERSHEIMER, M.D.

BOSTON

HIGH AND LOW INTRAOCULAR PRESSURE IN RELATION TO RETINOPATHY

During my studies on anatomic changes of the optic nerve associated with general hypertension, I examined the eyes of a patient aged 42 who was admitted to a well known hospital four times in one and one-half years because of chronic glomerulonephritis with increased blood pressure and uremic attacks. On each new admission the nonprotein nitrogen of the blood was at a higher level, the last time being as high as 507 mg. of urea and 9.1 mg. of uric acid per hundred cubic centimeters. In spite of this enormous elevation of nonprotein nitrogen and the high blood pressure, ophthalmoscopic examination revealed no spots or hemorrhages in the retina; even later, examination of many anatomic slides showed only one tiny group of "ganglioform nerve fibers." On the other hand, one could follow the development of a glaucomatous (from a physiologic) cupping during life, and after the patient died this was demonstrated histologically. Elschnig, one of the greatest authorities on the pathologic anatomy of glaucoma, to whom I sent the slides, confirmed the observation of glaucomatous cupping in both eyes. Although, to my regret, tonometric measurements were lacking in the record, it is probable that the intraocular tension, too, was high. Thus, there was the combination of high blood pressure, an unusually large amount of nonprotein nitrogen in the blood, primary glaucoma and absence of retinopathy. The problem arises: Had the glaucoma anything to do with the absence of retinopathy? I am inclined to answer "yes." As far as I can learn, nothing is known about such a relation. By chance, however, I found 2 interesting observations in the literature. Grafe,[1] in 1924, described briefly a case of retinitis diabetica in one eye which gradually diminished with the increase of intraocular tension in this eye; the other eye was glaucomatous for a long time, and in this eye diabetic retinitis never developed. Schieck,[2] in 1930, mentioned that retinitis albuminurica disappeared when glaucoma developed in the same eye. Both authors merely noted these observations but did not give them any serious consideration.

I personally saw a patient several months ago who probably belonged to this group. A Negress aged 79 had arteriosclerosis and general hypertension (blood pressure, 250 systolic and 160 diastolic). In September 1940 examination at the Massachusetts Eye and Ear Infirmary revealed pronounced, long-standing arteriosclerotic retinopathy with failing vision in both eyes and normal tension. I saw the patient for the first time on August 19, 1942, in the ophthalmic outpatient department of the Boston City Hospital. For six days she was totally blind in both eyes. She could not say whether she could see formerly with one or with both eyes. The right eye was very hard (tension, 65 mm. [Schiøtz]). The cornea was hazy. Only indirect ophthalmoscopic examination was possible, and this showed pronounced glaucomatous cupping but no retinal spots. The blind left eye had normal tension (14 mm. [Schiøtz]). The disk showed partial pallor; there were marked retinopathy and endovascular change. Several days later the tension in the right eye had been reduced almost to normal by miotics; the haziness of the cornea had disappeared, and I was able to see the fundus by direct ophthalmoscopic inspection. There were also many endarteritic vessels in this eye and numerous fine, yellowish spots in the retina, but, in contrast to the left eye, the latter were minute and in no way as striking as the brilliant whitish spots in the retina of the left eye. The difference in the appearance of the retina of the two eyes was notable in spite of the endovasculitis in both, and it is possible that the high tension in the right eye was responsible.

Read at a meeting of the New York Society for Clinical Ophthalmology, April 5, 1943, and, in enlarged form, before the Section of Ophthalmology of the College of Physicians of Philadelphia, Jan. 20, 1944.

1. Grafe, E.: Die Bedeutung der Insulintherapie des Diabetes für die Ophthalmologie, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **44**:53, 1924.

2. Schieck, F.: Die Erkrankungen der Netzhaut, in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 5, p. 425.

I owe the following observation to Dr. David Cogan, head of the Howe Laboratory of the Massachusetts Eye and Ear Infirmary:

A man aged 82 was admitted on Sept. 3, 1940, with the complaint of difficulty in reading, which had been worse in the past two or three weeks.

Right Eye: The pupil reacted sluggishly to light. The cornea was not steamy. Ophthalmoscopic examination revealed considerable arteriosclerosis, with obstruction of veins at the arteriovenous crossings and many flame-shaped hemorrhages. The optic cup was not glaucomatous. Vision was 20/100, and tension was 14 mm.

Left Eye: The pupil of this eye was similar to that of the right eye. The cornea was steamy. There was marked glaucomatous cupping. No hemorrhages were seen. Vision was uncertain, and tension was 28 — mm.

October 16: The field of the right eye was constricted. Vision was 18/200 with the patient's own glass. Vision in the left eye was limited to ability to count fingers at 2 feet (60 cm.). Only part of the temporal field remained. No hemorrhages were seen in this eye. The blood pressure was 160 systolic and 80 diastolic.

November 5: Vision was unchanged. Ophthalmoscopic examination showed a condition similar to that in the previous examination. Tension was 15 mm. in the right eye and 26 mm. in the left eye.

In this case, too, a marked difference in tension in the two eyes was noted; in the right eye it was rather low, but it was elevated in the left eye. The presence of such deep glaucomatous cupping in the left eye is in favor of the assumption that the tension was temporarily even higher than that noted in the record. In the right fundus, which showed physiologic cupping, there were many retinal hemorrhages, caused by the obstruction of veins and the sclerotic condition of the arteries. In the left, or glaucomatous, eye no hemorrhages could be seen, although the vascular condition was apparently the same as that in the right eye.

Altogether, it appears that a high intraocular tension influences the extravasation into the retina and that retinopathy, such as albuminuric or diabetic retinitis, either diminishes or disappears when glaucoma develops or that such a retinopathy does not occur in an already glaucomatous eye. Of course further clinical experience is necessary to settle this point.

The logical corollary of such a concept is that a low intraocular tension produces a tendency to an especially pronounced extravasation from the retinal vessels, especially in cases of general hypertension. As far as I know, there do not exist statistics on intraocular tension in cases of retinopathy, but Dr. Cogan with whom I discussed this problem, showed me a list of such cases, in which he had been called in consultation and in which he had tested the intraocular tension as a routine. It was interesting that in most of them—not in all—the intraocular pressure was surprisingly low.

RELATION OF HEMORRHAGIC OR NONHEMORRHAGIC CHARACTER OF VENOUS OBSTRUCTION TO INTRAOCULAR TENSION

Not much attention has so far been paid to the statement that the intraocular tension is generally low in cases of retinal venous thrombosis. Moore [3] stated that in all but 1 of his 13 cases of total obstruction tension was lower than that in the healthy eye. Wessely [4] confirmed this statement from his own experience; he noticed this difference also in cases of thrombosis of a tributary vein. Wessely stated that this decrease of tension was probably due to narrowing of the retinal and uveal arteries.

Development of the venous thrombosis has of course nothing to do with the lowered intraocular tension; the question is, however, whether the extravasation of blood is facilitated by the diminished pressure outside the vessel wall.

In this connection, it is interesting to compare anatomically verified cases of retinal venous obstruction in which, as usual, the obstruction was primary and the glaucoma secondary with cases in which glaucoma existed at first and the obstruction developed later.

In 1913 Verhoeff [5] studied the effect of chronic glaucoma (following occlusion of the pupil, corneal ulcer, anterior staphyloma, cataract extraction or serous iritis) on the central retinal vessels in 39 cases. He showed that in every case there was endovasculitis of the central vessels and that there was frequently, especially in the central vein, complete, or almost complete, obstruction. The changes in the central vessels were essentially the same as those which have been described in the literature and which Verhoeff himself had observed in association with hemorrhagic glaucoma. He stated:

The almost complete absence of retinal hemorrhages in this series of cases is noteworthy. Thus, out of fifteen cases of complete or almost complete obstruction of the central vein, in only one case were they present in any significant number. In two other cases there were a few small hemorrhages, but it was doubtful if they were the result of the obstruction of the central vein.

Neither was edema of the retina prominent in any one case. According to Verhoeff, the infrequent occurrence of retinal hemorrhage was probably due in some cases to the extensive

3. Moore, F.: Retinal Venous Thrombosis: A Clinical Study of Sixty-Two Cases Followed over Many Years, London, George Pulman & Sons, Ltd., 1924.

4. Wessely, K.: Ueber die Prognose der Thrombose der Centralvene, Klin. Monatsbl. f. Augenh. **95**:398, 1935.

5. Verhoeff, F. H.: The Effect of Chronic Glaucoma on the Central Vein Vessels, Arch. Ophth. **42**: 145, 1913.

involvement of the artery and in others to the slowness of the process, which allowed for the establishment of adequate collateral circulation.

An article by Salzmann [6] on "glaucoma and retinal circulation," to which I [7] referred in a previous paper, also has a relation to the topic under consideration. He studied the anatomic condition of the central vein and the retina in 80 eyes which were enucleated because of absolute glaucoma. In most cases the condition was of too long standing for one to evaluate which had come on first, the glaucoma or the venous obstruction. In a few cases, however, the sequence was fairly certain. In cases 42 and 43, patients with general hypertension had typical thrombosis of the central vein with numerous retinal hemorrhages and exudates, and several weeks later glaucoma developed. In contrast to this usual course of events, there were other cases in which the glaucoma preceded venous obstruction. For example, in case 49 a woman aged 64 with glaucoma showed a severe and progressive visual disturbance four years after the appearance of glaucoma. Ophthalmoscopic inspection revealed tortuous veins in the area of deep glaucomatous cupping, but neither ophthalmoscopic nor histologic examination showed any hemorrhages or exudates in the retina. Autopsy revealed absolute obstruction of the central vein. In case 50 a man aged 64 had traumatic luxation of the lens with subsequent glaucoma. Enucleation was performed one year after the injury. Histologically, there were great changes in the venous vessels, especially in the superior retinal vein, but no hemorrhages in the retina could be seen. Salzmann was in favor of the idea that the absence of hemorrhages was due to newly formed collaterals in the retina. Also, Scheerer [8] made two observations of interest in connection with this question. In his case 1 the glaucoma was primary and the thrombosis of the central vein secondary. The latter condition could be diagnosed only anatomically, after enucleation, for during life no retinal hemorrhages were visible. Case 4 was similar in this respect. But in case 14 there was glaucoma simplex, with eventual development of retinal hemorrhages. Enucleation was performed one year after the appearance of the ophthalmic trouble. Histologic study showed that the central artery and the central vein were almost completely obstructed.

The studies of Verhoeff, Salzmann and Scheerer were made chiefly from an anatomic point of view; the clinical data given were generally brief, or the situation was so complicated that most of the cases could not be used in the present study. The observations mentioned, however, seem to support the view that intraocular pressure often has a bearing on the presence or absence of retinal hemorrhage. It may be added that Coats,[9] in his exhaustive work on obstruction of the retinal vein, pointed out that in his experience the sequence of glaucoma first and obstruction of the retinal vein later is extremely rare. He encountered only 1 case, and that was doubtful. In Coats's opinion, only cases with retinal hemorrhages were recognized as full fledged instances of venous thrombosis. Thus, he apparently missed the cases without hemorrhages.

In 1905, Harms [10] had stated: "It is a well known fact, that the opportunity of observing venous obstruction in an eye with primary glaucoma is rare."

PAPILLEDEMA AND HEMORRHAGES IN EYES WITH DECREASE OF INTRAOCULAR TENSION

Another condition undoubtedly related to lowering of the intraocular pressure must be discussed. This is the papilledema which occurs sometimes in eyes with very low pressure (after penetrating wounds or in disease), and which disappears when the intraocular tension becomes normal. It is thought to be of purely mechanical origin, like the papilledema associated with high intracranial pressure (Behr,[11] Duke-Elder [12] and others). It could be proved anatomically that no inflammatory factor is involved. It is not easy to explain why such edema develops in the disk only, and why it occurs only in certain cases. The diminished ocular tension is apparently the most important, but not the only, etiologic factor. There is edema, but no hemorrhages or exudative patches, probably because neither the content nor the walls of the vessels are pathologic. The transudation is facilitated by a vascular dilation, which follows the sudden fall of intraocular pressure. According

6. Salzmann, M.: Glaukom und Netzhautzirkulation, Berlin, S. Karger, 1933.

7. Igersheimer, J.: Ist ein Glaukom imstande die Transsudation aus den Netzhautgefässen zu beeinflussen? Ophthalmologica **97**:146, 1939.

8. Scheerer, R.: Zur pathologischen Anatomie der Veränderungen der Netzhautzentralgefässe bei der sogenannten Thrombose der Zentralvene und Embolie der Zentralarterie, Arch. f. Ophth. **110**:292, 1922; **112**:206, 1923.

9. Coats, G.: Der Verschluss der Zentralvene der Retina, Arch. f. Ophth. **86**:340, 1913.

10. Harms, C.: Anatomische Untersuchungen über Gefässerkrankungen im Gebiete der Arteria und Vena centralis retinae, Arch. f. Ophth. **61**:1, 1905.

11. Behr, C.: Ueber die im Anschluss an perforirende Bulbusverletzungen auftretende Stauungspapille, Klin. Monatsbl. f. Augenh. **50**:56, 1912.

12. Duke-Elder, W. L.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1941, vol. 3.

to Sobansky's studies, cited by Lauber,[13] the relation between the pressure in the retinal arteries and that in the retinal veins, seems undisturbed.

Kyrieleis [14] stated that there is no doubt the papilledema in eyes with low intraocular pressure is mechanical, but that increased transudation through the vessel walls as a cause of the papilledema is of little importance, since under such circumstances diffuse edema of the retina would necessarily develop. His explanation is as follows: 1. The lymph fluid of the eye normally has some outflow into the optic disk, and this outflow is favored by intraocular pressure. If the intraocular tension is low, there will be stagnation of the lymph in the disk. 2. The normal tissue pressure in the optic nerve between the disk and the lamina cribrosa is determined by the blood pressure and the intracranial pressure from the intravaginal space. If the intraocular tension is low, the anterior part of the optic nerve may protrude like a hernia.

It is noteworthy, also, that under the reverse conditions, namely, acute inflammatory glaucoma, hyperemia with slight edema of the disk, sometimes combined with retinal hemorrhages, may occur. In my opinion the reason that this hyperemia and edema occur under such different circumstances, with especially low and with especially high intraocular pressure, is as follows: In the case of extreme hypotony the lowered pressure is primary and the papilledema secondary, whereas in the case of acute glaucoma the great influx of blood into the ocular vessels and the increased transudation are probably primary, with a resulting high intraocular tension. As will be remembered, Friedenwald,[15] in a histologic study of acute glaucoma, noted the presence in all cases of edema of the ciliary body, which pressed the iris toward the angle of the anterior chamber.

If the intraocular tension is high and the vessel walls are diseased and friable because of arteriosclerotic changes, sudden diminution of the pressure may have the tragic result of an uncontrollable hemorrhage, as is well known.

Only a short time ago, I found another statement in line with the concept under discussion. Parker [16] gave evidence of the fact that even papilledema caused by increased intracranial pressure has some relation to the intraocular pressure. When both eyes have equal tension, papilledema develops to an equal degree in the two eyes, but if and when the tension in one eye is lower than that in the other eye, papilledema generally develops first in the eye with the lower tension. Still more impressive were experimental studies of Parker on dogs and monkeys. The intraocular tension of one eye was reduced by trephination. After recovery from the effects of the operation, a second ophthalmoscopic examination was made, and, again, the tension was recorded. Then artificial intracranial pressure was induced by different means on the side opposite the eye in which the tension had been reduced. Each time papilledema developed first in the eye with the reduced tension. Bordley [17] confirmed the clinical statement of Parker and added an interesting experience. In a case of tumor of the brain one eye suffered from glaucoma. In this glaucomatous eye choked disk did not develop.

PRESSURE OUTSIDE THE VESSEL WALL IN PHYSIOLOGY OF TRANSUDATION

Passage of fluid through the capillary wall is frequently discussed in general physiology. The extensive studies of Krogh and Lewis have shown that the capillary vessels are independently contractile. The permeability of the capillary wall varies within wide physiologic limits; at one moment the capillary walls in a given area may be almost totally impermeable to colloidal substances, and shortly afterward, under the influence of lack of oxygen or of some other stimulus, the permeability may be greatly increased, so that blood plasma escapes readily into the tissue spaces. Such a change in permeability is characteristic of capillaries, whereas it is absent in arteries (Ebbecke [18]). Vasodilation increases, and vasoconstriction reduces, capillary permeability. According to Landis,[19] important factors controlling the movement of fluid through a membrane are as follows: (1) total area of filtering surface available; (2) properties, particularly permeability, of the membrane itself, and (3) pressures exerted on the fluids inside and outside the membrane.

13. Lauber, H.: The Formation of Papilledema, Arch. Ophth. **13**:733 (May) 1935.

14. Kyrieleis, W.: Ueber Stauungspapille, Arch. f. Ophth. **121**:560, 1929.

15. Friedenwald, J. S.: The Pathogenesis of Acute Glaucoma: Clinical and Pathologic Study, Arch. Ophth. **3**:560 (May) 1930.

16. Parker, W. R.: (*a*) The Relation of Choked Disc to Intraocular Tension: A Clinical Study of Six Cases, Ann. Ophth. **20**:715, 1911; (*b*) The Relation of Choked Disk to the Tension of the Eye, J. A. M. A. **67**:1053 (Oct. 7) 1916; (*c*) The Mechanics of Papilledema, Tr. Am. Acad. Ophth. **29**:77, 1924; (*d*) The Relation of the Tension of the Eyeball to the First Appearance of Papilledema, A. Research. Nerv. & Ment. Dis., Proc. **8**:256, 1929.

17. Bordley, J., in discussion on Parker,[16b] p. 1056.

18. Ebbecke, U.: Gefässreaktionen, Ergebn. d. Physiol. **20**:401, 1923.

19. Landis, E. M.: The Passage of Fluid Through the Capillary Wall, in Harvey Lectures, 1936-1937, Baltimore, Williams & Wilkins Company, 1937, p. 70.

The permeability of the vessel wall, the blood pressure and the osmotic pressure are thought to be the principal factors in such a transudative process. The pressure outside the vessel wall is not often mentioned, for generally the circumstances are such that it does not play a great role. Edema, especially the experimental wheal, is the phenomenon by which the migration of fluid through the vessel wall has been chiefly studied experimentally. In these studies it was proved that the pressure outside the vessel wall may be of significance. Before the wheal itself develops, there is capillary dilation; the necessary increase of capillary permeability does not occur without such a dilation. If a wheal develops quickly, the capillary dilation will be counteracted by the pressure of the edema (Ebbecke[18]). Lewis,[20] in his experimental studies on vascular reactions, stated that the wheal normally following a heavy stroke may be prevented by pressure from outside, such as that exerted by an armlet. When a small wheal develops under a pressure capsule, it is small because the outside pressure checks the rate of blood flow in the affected vessels. In his Harvey Lecture, Landis[19] came to the following conclusions:

> Apparently extravascular fluid distends the tissue spaces by developing a tissue pressure which at the same time diminishes progressively the effectiveness of a given capillary pressure in producing further filtration. The physiological importance of this tissue pressure is probably considerable.

The same author, in summarizing the factors concerned in the pathogenesis of edema, differentiated primary factors favoring formation of edema, such as elevated capillary pressure, lowered osmotic pressure to colloids, damage to the capillary wall and lymphatic obstruction, and contributory factors. The most important contributory factor is, according to Landis, a low tissue pressure, but he added:

> . . . though the resistance of the tissues can delay, it cannot prevent the appearance of edema when venous congestion, capillary damage, or reduced colloid osmotic pressure favor filtration continuously over long periods of time.

COMMENT

From this brief excursion into general physiology, it can be seen that the pressure outside the vessel wall is recognized as a factor which may influence the transudation through the wall. Low external pressure favors, and high external pressure reduces or hinders, the transudation. Generally this factor does not play a great role, because there rarely occurs a condition in which pressure outside the vessels becomes very high and localized, unless applied artificially by an armlet or other means. The situation is, of course, different in closed capsules, like the eyeball or the skull.

It is old knowledge that sudden lowering of the intraocular tension is often followed by dilation of the intraocular vessels, and even by a severe hemorrhage, if the vessel walls are abnormally permeable. The intraocular pressure, which is of course the pressure outside the vessel wall, seems to be of greater importance for the transudative mechanism in general than has hitherto been thought. If further studies confirm the aforementioned experiences, it will be established that intraocular tension influences the development and the type of exudation occurring with retinopathies. Low tension seems to facilitate such exudation, while glaucoma may reduce its extent, prevent it or cause the exudate to disappear entirely. Similarly, the hemorrhages associated with obstruction of a retinal vein generally occur in an eye with low tension and may be entirely absent in a primarily glaucomatous eye, even though the retinal vein is completely obstructed. It is interesting that the papilledema, in its development and sometimes in its degree, is also influenced by the intraocular pressure. This is valid not only for papilledema caused by intraocular hypotonic conditions (penetrating wound; iridocyclitis) but for the papilledema following increased intracranial pressure (Parker). Glaucoma prevented the development of papilledema in a case of cerebral tumor (Bordley). In retinopathy, as well as in thrombosis of the central vein, the situation is of course often complicated by a high arterial and capillary pressure, by a change in the protein content of the blood and by pathologic changes in the vessel wall. Thus, it would not be surprising if a high intraocular tension is sometimes insufficient to counteract transudation. Landis expressed similar views concerning the limitations of tissue pressure when other factors are too powerful.

Altogether, it seems of interest that clinical pictures which are so different in their origin and clinical aspects as retinopathies, venous obstruction and papilledema may have one factor in common, namely, that of being influenced by the intraocular pressure. It is interesting, also, that clinical ophthalmologic facts confirm the physiologically recognized significance which the external pressure has in the passage of contents of the blood through vessel walls, and that perhaps nowhere in the body are the conditions for proving this significance so ideal as in the globe.

Whether this whole concept can be utilized practically is a question for the future.

20. Lewis, T.: Vascular Reactions of the Skin to Injury, Heart **11**: 119,, 1924.

SUMMARY

It is well known that the hydrostatic pressure, as well as the condition of the capillaries (constriction, dilation and permeability of the wall), the content of the blood and the resistance to the outflow of blood and lymph from the globe are the main factors which, if pathologically altered, may produce an increased passage of fluids and corpuscles through the vessel walls of the retina. It was not realized until now that, besides these factors, the intraocular pressure seems to play a certain role in these transudative phenomena. A low tension facilitates transudation; a high tension may prevent exudation in eyes predisposed to retinopathy or in cases of obstruction of retinal veins. Such phenomena in the eyeball serve as especially good evidence for the general physiologic assumption that the pressure outside the vessel wall is of significance for the passage of fluid through the capillary wall.

636 Beacon Street.

DEFECTS IN VISUAL FIELDS PRODUCED BY HYALINE BODIES IN THE OPTIC DISKS

C. WILBUR RUCKER, M.D.
ROCHESTER, MINN.

The presence of hyaline bodies in the optic disks occasionally produces an appearance that may simulate that of chronic papilledema with secondary optic nerve atrophy. When the glistening bodies are large and lie near the surface, they cause little difficulty in diagnosis, but when they are small and lie buried so deeply within the nerve substance that they are scarcely visible, they may cause a fulness that is difficult to explain. In fact, unless they are looked for specifically, they may pass unnoticed.

Not only is the appearance of the optic disks misleading, but field defects are encountered rather frequently in this condition, either in the form of peculiar contractions or of arcuate scotomas. Such defects may confuse the diagnostician still further. This impairment of vision was mentioned by Reese [1] in his excellent review of the subject in 1940.

In general, however, little attention has been paid to the associated visual defects; therefore, a consideration of this phase of the subject appears to be timely. I have reviewed the cases that I have seen during the past few years, and from the group in which the perimetric fields were plotted I have selected a few diverse types. They largely demonstrate how variable may be the defects.

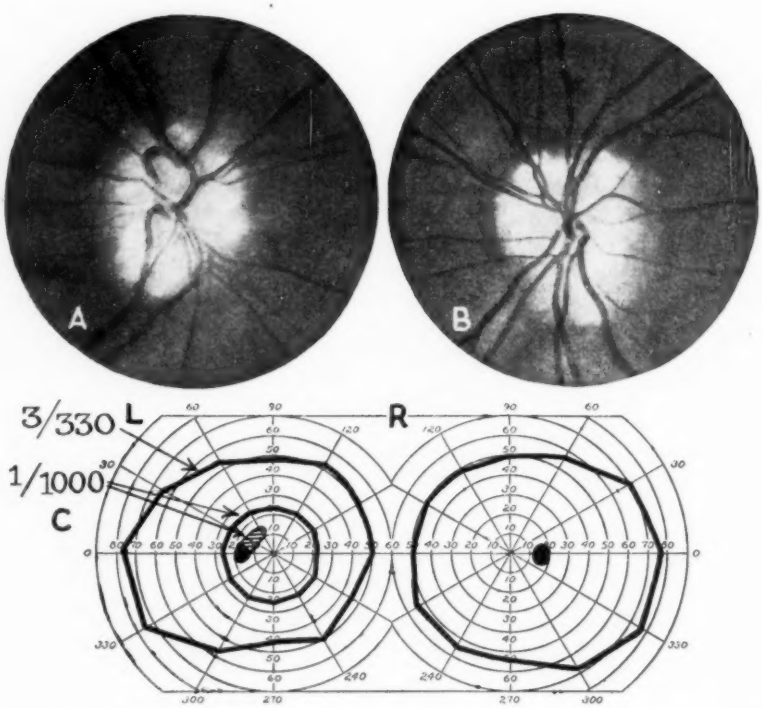


Fig. 1 (case 1).—(*A*) right fundus; (*B*) left fundus; (*C*) visual fields. Vision was 6/10 in each eye.

REPORT OF CASES

CASE 1.—A woman aged 29 complained of nervousness, increased appetite and loss of weight. These were found to be due to exophthalmic goiter, and subtotal thyroidectomy was performed. There were no symptoms refer-

From the Section on Ophthalmology, the Mayo Clinic.

1. Reese, A. B.: Relation of Drusen of the Optic Nerve to Tuberous Sclerosis, Arch. Ophth. **24**:187-205 (July) 1940.

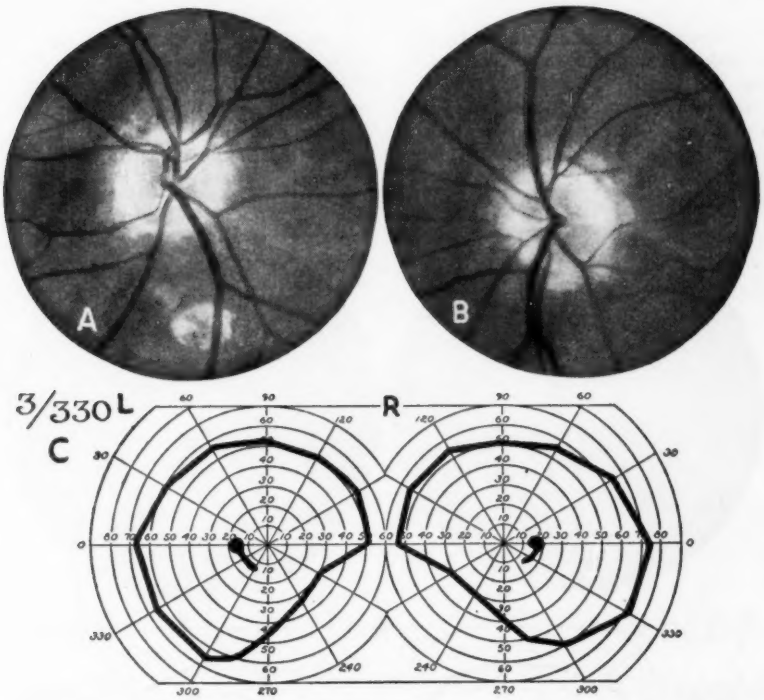


Fig. 2 (case 2).—(*A*) right fundus; (*B*) left fundus; (*C*) visual fields. Vision was 6/6 in the right eye and 6/10 in the left eye.

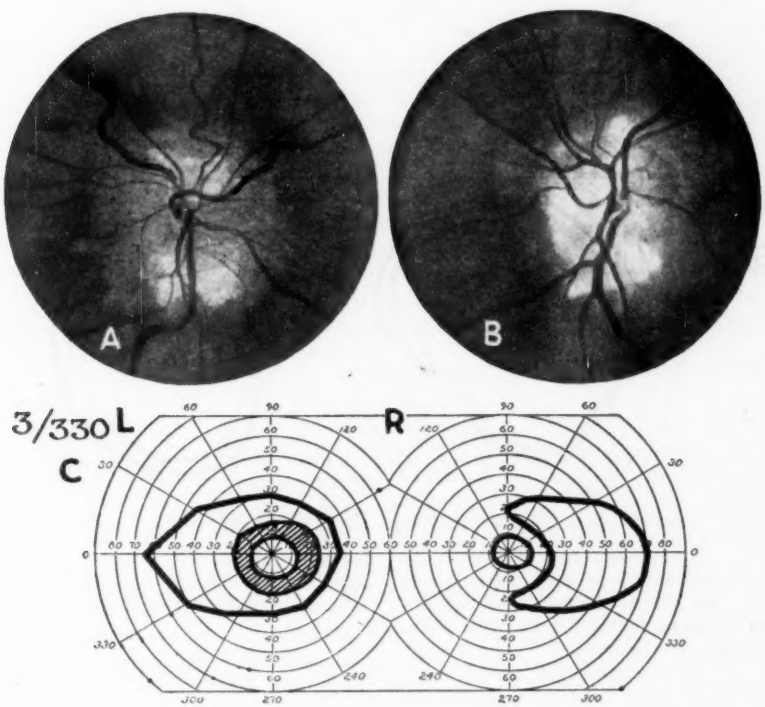


Fig. 3 (case 3).—(*A*) right fundus; (*B*) left fundus; (*C*) visual fields. Vision was 6/7 in the right eye and 6/10 in the left eye.

able to the eyes. Neurologic examination was not performed.

Ophthalmoscopic examination disclosed large hyaline masses in both optic disks. The right was elevated 2 D. and the left 1 D. (fig. 1 *A* and *B*).

When the visual fields were plotted, it was found that the normal blindspot of the right eye was enlarged and that a small arcuate scotoma extended upward from the blindspot of the left eye (fig. 1 *C*).

of the pain, and abdominal hysterectomy was performed. Neurologic examination disclosed no abnormalities except absence of deep tendon reflexes in the legs. There were no visual symptoms.

On ophthalmoscopic examination, hyaline masses were seen on both optic disks; the right disk was elevated 2 D. and the left 1 D. (fig. 3 *A* and *B*).

Plotting of the fields of vision disclosed a dense ring scotoma in the field of the left eye and a similar ring,

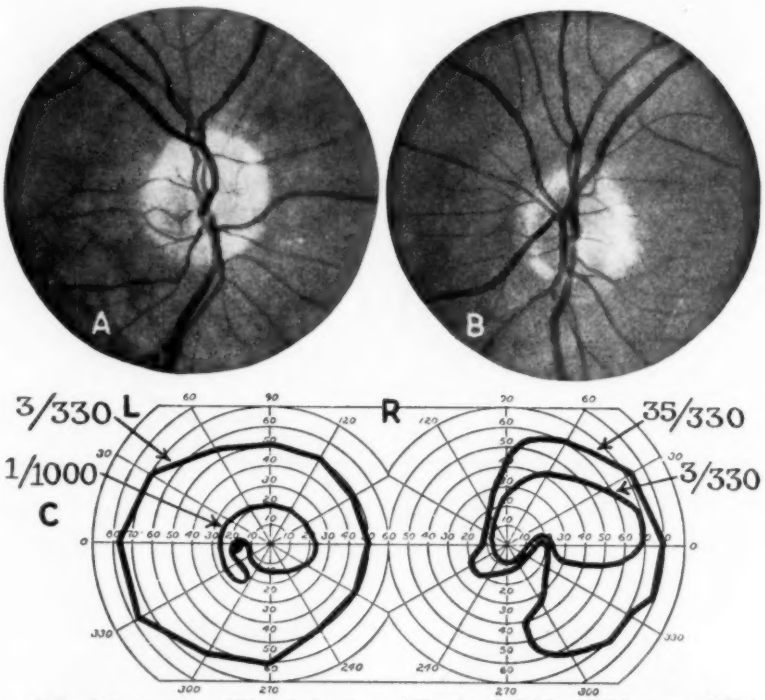


Fig. 4 (case 4).—(*A*) right fundus; (*B*) left fundus; (*C*) visual fields. Vision was 6/7 in the right eye and 6/6 in the left eye.

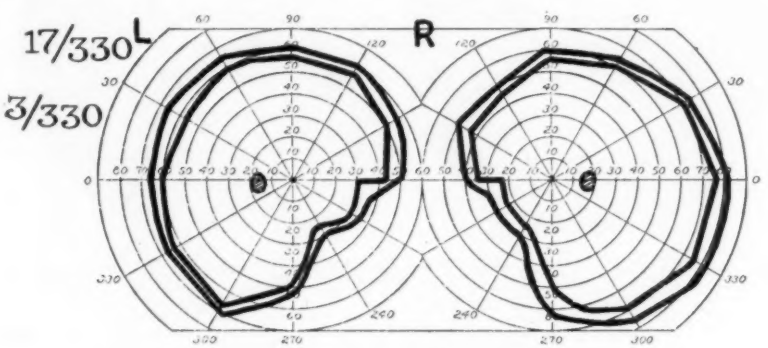


Fig. 5 (case 5).—Visual fields. Vision was 6/6 in each eye.

Case 2.—A woman aged 46 complained of headaches. For many years they had occurred every few months, but had become more frequent during the past four years and constant for the past four months. The neurologic diagnosis was "involutional change in migraine."

On both optic disks there were piles of hyaline bodies (fig. 2 *A* and *B*).

Plotting of the visual fields revealed binasal contraction and small, dense arcuate scotomas pointing downward (fig. 2 *C*).

Case 3.—A woman aged 49 complained of pain in the back. Large uterine fibroids were found to be the cause which had broken through to the nasal periphery, in the field of the right eye (fig. 3 *C*).

Case 4.—A man aged 39 complained of headache of six weeks' duration. His family physician had plotted his visual fields and, on finding a defect, had urged him to go to a large clinic for neurologic and ophthalmic examinations.

Ophthalmoscopic examination of the right optic disk disclosed several hyaline bodies, which gave it a pale color, and at the nasal margin of the left disk there were two deep bodies of a similar nature (fig. 4 *A* and *B*).

The visual fields contained arcuate scotomas; the one in the right eye had broken through to the nasal periphery (fig. 4 *C*).

CASE 5.—A girl aged 15 years had consulted her local oculist because for a year she had been having occasional headaches and thought she might need glasses. On examination of her eyegrounds, he suspected the presence of chronic papilledema, and his suspicion was not allayed when he plotted the visual fields and found binasal contraction. Because of these findings, he asked her to seek further advice at a large clinic. Extensive clinical and laboratory tests and a neurologic examination failed to disclose any abnormalities.

Many hyaline bodies were seen in the optic disks on ophthalmoscopic examination. The fundi were not photographed.

Binasal contraction was demonstrated by plotting the visual fields (fig. 5).

COMMENT

These cases illustrate the usual varieties of defects in the visual fields caused by hyaline bodies on the optic disks. There may be enlargement of the normal blindspots and arcuate scotomas, either in the form of small fingers or of large arms breaking through to the periphery, or there may be peripheral contraction, which most often is below and on the nasal side.

TUBEROUS SCLEROSIS ASSOCIATED WITH TUMOR OF THE OPTIC DISK (PHACOMA)

MAJOR EARL A. GLICKLICH, MAJOR ABRAHAM SCHULTZ
AND
COLONEL JULIEN E. BENJAMIN
MEDICAL CORPS, ARMY OF THE UNITED STATES

We are reporting a case of tuberous sclerosis with the characteristic syndrome of adenoma sebaceum, epilepsy and mental deficiency, associated with tumor of the optic disk (phacoma).

REPORT OF CASE

History.—An American soldier aged 22, unmarried, was first seen in December 1942. He was admitted from an overseas hospital with a transfer diagnosis of tuberous sclerosis and epilepsy of the grand mal form.

The patient had been inducted into the Army on Feb. 10, 1942 at Fort Bragg, N. C. Several weeks later he had had an epileptiform seizure from which he promptly recovered. In March 1942 he had pneumonia, for which he was hospitalized one month.

He was sent overseas in July 1942. He felt well except for a residual dry cough which had persisted since the attack of pneumonia. The cough became more frequent at night, although it was nonproductive. On Aug. 28, 1942 the patient was admitted to an overseas hospital because the cough had become progressively worse. During hospitalization the cough improved. On further investigation of his epileptiform seizures, it was suspected that this patient had tuberous sclerosis, as manifested by a familial history of epilepsy and the presence of cutaneous lesions and of vestiges of congenital anomalies. The patient was therefore returned to the United States for further observation and treatment.

The patient was born April 28, 1919. He was a full term baby and had a normal birth. He talked early, walked at the age of 14 months and teethed at normal age. He had measles, mumps and whooping cough in childhood. He entered school when he was 7 years old and completed the fourth grade at the age of 14. He learned to read and write but never learned arithmetic. In his youth he worked on farms with his parents, where he was able to perform simple chores.

His first epileptiform seizure occurred at the age of 7, and since then he had had four similar attacks. The last seizure occurred shortly after induction, as previously mentioned. The patient stated that the onset of these seizures was preceded by dizzy sensation and loss of vision, followed by loss of consciousness. On regaining consciousness he noted weakness and nausea, after which he fell into a deep sleep. The patient did not recall biting his tongue or injuring himself during any of these seizures.

Family History.—Investigation of the family by the Red Cross revealed that the father was 63 years old and slightly deaf. He had high blood pressure and had a cutaneous condition similar to that present in the son, involving the face and nose. He impressed one as being hopelessly limited both physically and mentally. The mother was 60 years old and presented no cutaneous changes. She showed more initiative than the husband, though she was illiterate. Two sisters of the patient were living and well. One of the sisters had a condition of the skin of the face similar to that of the patient. The patient had four brothers, of whom only the oldest was living. He was 39 years old, had had epilepsy for four years as a child and also had cutaneous lesions on the face resembling those of the patient. He had not gone beyond the third grade of grammar school. He had been crippled in an accident and appeared to be definitely mentally retarded. Three brothers had died, one at the age of 10 months, of colitis, another at the age of 3 years, of pneumonia and the third at the age of 20 years, of undetermined cause. The last-mentioned brother, who had not advanced beyond the second grade of grammar school, had had epileptic attacks which started at the age of 4 years and continued until his death. He was always an invalid and was considered to be of low mentality.

Physical Examination.—The patient was 70 inches (178 cm.) in height, weighed 165 pounds (74.8 Kg.) and did not appear acutely ill. The pharynx and teeth were normal. There was no significant adenopathy. Examination of the heart, lungs and abdomen revealed no abnormal conditions. Neurologic examination gave essentially normal results. The blood pressure was 110 mm. systolic and 70 mm. diastolic.

Dermatologic Examination.—Definite lesions of adenoma sebaceum (fig. 1) were present on the face, being characterized by areas the size of a pinhead to that of a split pea distributed over the nose, cheeks and nasolabial folds. The lesions were yellowish and pinkish, discrete, soft and moderately compressible, and the superficial capillaries in the affected region were generally telangiectatic. A hard, whitish cystic growth was present just below the right internal canthus. Similar lesions were also present behind the ears. The skin of the trunk and extremities was normal in appearance and texture.

Ocular Examination.—The patient had no symptoms referable to his eyes. Vision was 20/20 in each eye, and small print could be read with ease. The eyes were essentially normal with the exception of the right fundus. In the latter an oval, glittering, gray mulberry-like growth occupied the inner half of the disk and projected over its nasal margin. The mass was elevated 2 diopters. The surface of the lesion consisted of closely packed nodular cysts, over which the retinal vessels coursed. A small, superficial, striated area of hemorrhage was present near the inferior pole of the tumor (fig. 2). The visual fields were normal except for an enlargement of the physiologic blindspot in the right eye, which was extended temporally to twice the normal size.

Mental Examination.—The patient was orderly, friendly and cooperative. His speech was spontaneous, coherent and relevant. His mood was normal, and no

delusions or hallucinations were present, but he was only partially oriented. A Kent emergency test gave an intelligence quotient of about 65. The Stanford-Binet formal test revealed a quotient of 70 and a mental age of 11 years and 2 months. The clinical impression of the psychiatrist was that the patient was a borderline mentally defective person with defects most evident in the fields of memory, retention and recall.

Laboratory Examination.—The sedimentation rate, the serologic reactions of the blood, the amount of nonprotein nitrogen in the blood, the sputum, and the cellular elements of the blood (by complete blood count) were all within normal limits, except the leukocytes, which were slightly increased in number. The urine contained numerous white and red blood cells, with occasional hyaline casts, on several occasions.

Roentgenographic Observations.—Roentgen examination of the chest revealed no abnormality of the heart or lungs. The calvarium was of average thickness. No evidence of abnormal calcification was present. The sella turcica appeared normal, and the pineal calcification occupied its usual position. An encephalogram revealed slightly dilated ventricles with irregularities of their walls. No abnormalities were noted in the long bones with the exception of an old ununited fracture of the right ulnar styloid process. Intravenous and retrograde pyelography revealed no abnormal conditions.

Histologic Examination of Skin (Made by Lieut. Col. Robert Fienberg).—Numerous small and large sebaceous glands were found in the corium. No true neoplasia was evident. The ducts were dilated and filled with keratin. A few dilated blood vessels were seen. Scattered lymphocytes were present in the corium, and a few pigmented phagocytes were present just below the basal cells of the epithelium.

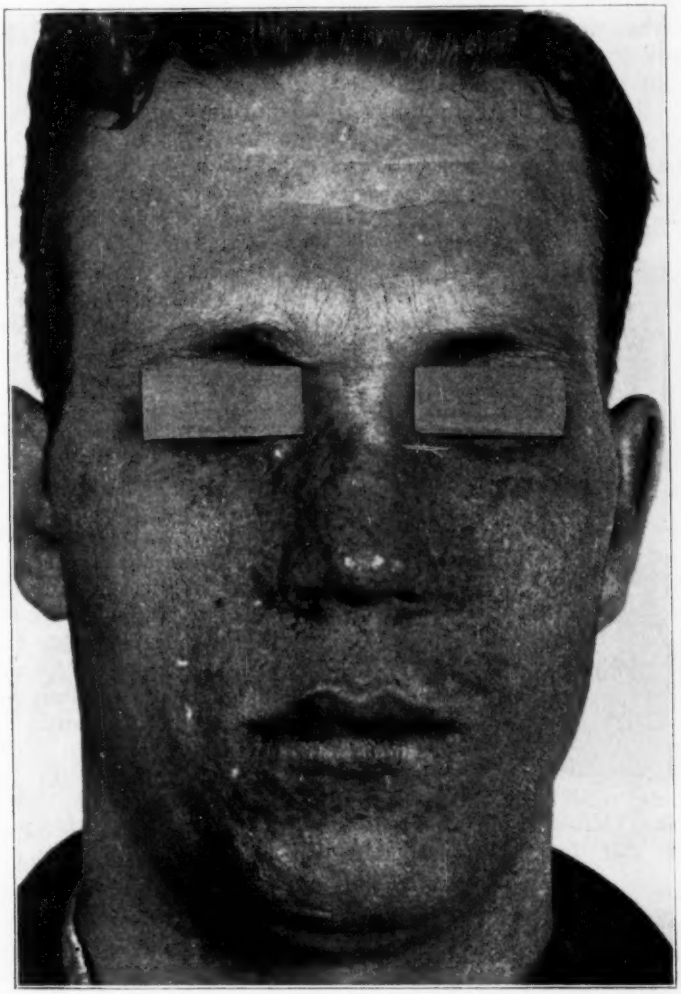


Fig. 1.—Photograph of a patient with tuberous sclerosis showing the associated cutaneous lesions.

Course in the Hospital.—No epileptiform seizures were observed during the period of hospitalization. It was established that the syndrome of tuberous sclerosis, comprising mental deficiency, epilepsy and adenoma sebaceum, was present in the patient. In addition, a tumor of the optic disk was present in the right fundus. It was suspected that tumors were also present in the ventricles of the brain because of the conditions described in the report on an encephalogram from the overseas hospital. Renal involvement was suspected because of the numerous red and white cells occasionally present in the urine, though retrograde and intravenous pyelo-

grams revealed no abnormality. Because of the conditions discovered the patient was separated from the military service and escorted home.

COMMENT

Adenoma sebaceum is one of the cutaneous manifestations associated with the syndrome known as tuberous sclerosis. The other manifestations of the disease are epilepsy and low grade mentality. Evidence of the condition is usually present in one or more members of the family. Autopsy reveals interesting changes in the brain in the nature of nodular sclerosis of the cerebral cortex with hard, tiny, potato-like tumors, from which the name of the condition is derived. Other congenital anomalies have been reported involving various organs, such as the kidneys, heart, liver, gastrointestinal tract and retina. The association of epilepsy with this condition is of interest. It is possible that in many cases of "idiopathic epilepsy" tuberous sclerosis is the pathologic basis.

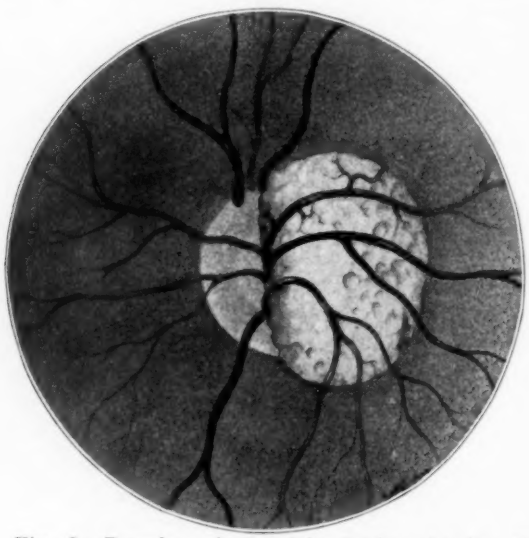


Fig. 2.—Drawing of the right fundus showing the tumor of the optic disk. (Miss Betty Foss, of the Massachusetts Eye and Ear Infirmary, made this drawing.)

Phacoma of the optic disk is particularly interesting in this case because of the extreme rarity of reports of this condition in the American literature. Though Bourneville had already established the syndrome of tuberous sclerosis in 1880, the associated retinal lesions were not recognized until 1921, when van der Hoeve [1] first described them in reports on 6 patients suffering from this disease. He called the lesions *Phakomata* from the Greek word *phakos,* meaning "mother spot," believing that they arose from embryonic cell rests. Since then relatively few retinal tumors have been described. Critchley and Earl [2] found only 1 phacoma in their series of 29 cases of tuberous sclerosis. In a comprehensive review of the literature by Messinger and Clarke [3] 18 more cases of retinal tumors in tuberous sclerosis were added. In 5 of the cases there were phacomas of the optic disk as well as the retina. To this group the authors added their own case of tumor of the nerve head with descriptions of the ophthalmoscopic and histologic observations. Other instances of phacoma of the retina have been described by Gifford,[4] Bloch and Grove [5] and Koch and Walsh.[6] On the basis of retinal changes Givner [7] diagnosed tuberous sclerosis in a 5 month old infant with convulsions. The diagnosis was subsequently confirmed by an encephalogram which showed the "candle droppings" of the ventricle.

Though phacomas occur less frequently in tuberous sclerosis than the cutaneous lesions or the mental changes, it must be remembered that changes in the fundus may sometimes be the sole manifestation of this syndrome. Under such circumstances tuberous sclerosis must be differentiated from the allied conditions in which retinal phacomas occur. These conditions include: (1) von Recklinghausen's neurofibromatosis, which affects the skin and cranial nerves; (2) von Hippel's retinal and Lindau's cerebral angiomatosis; (3) Sturge-Weber's syndrome (angiomatosis cerebri), characterized by buphthalmos, enlargement of the cerebral vessels and nevus flammeus, and (4) retinal glioma in infants. The changes in the fundus in tuberous sclerosis are polymorphic; the phacomas may be single or multiple, flat or raised, and there may be various pigmentary changes in the retina. About one fifth of the retinal phacomas are confined to the optic disk, as in this case. The most typical lesion is an elevated, gray, mulberry-like growth, such as was present in the fundus of this patient.

The origin of tumors of this type is still not certain, though they are generally recognized to be primarily ectodermal malformations, consisting of glial tissue and undifferentiated elements, including mesoderm, initiated by developmental and hereditary factors.

1. van der Hoeve, J.: Retinal Tumors in Tuberous Sclerosis, Arch. f. Ophth. **105**:880, 1921.

2. Critchley, M., and Earl, C. J. C.: Tuberose Sclerosis and Allied Conditions, Brain **55**:311 (Sept.) 1932.

3. Messinger, H. C., and Clarke, B. E.: Retinal Tumors in Tuberous Sclerosis: Review of Literature and Report of a Case, Arch. Ophth. **18**:1 (July) 1937.

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CHALCOSIS LENTIS ASSOCIATED WITH TRAUMATIC LENTICONUS POSTERIOR

CAPTAIN EMANUEL ROSEN
MEDICAL CORPS, ARMY OF THE UNITED STATES

The chemical reaction of copper in the lens is one of the few ophthalmologic subjects which has not been overemphasized in the literature. Chalcosis lentis has been encountered frequently during the present war because of the employment of numerous alloys in armaments, some of which contain copper. The toxicity of copper within the globe is well known, and the production of a characteristic "sunflower" just below the anterior capsule of the lens has been regarded as one of the pathognomonic signs of chalcosis lentis.[1] The presence of the "sunflower" is frequently difficult to determine, and certain methods of examination should be employed to bring out this outstanding characteristic. A "sunflower" has been reported by Thiel as occurring in cases of hepatolenticular degeneration, but this lesion has not the appearance or the color of that associated with chalcosis lentis.

Since the "sunflower" occurs in the region of the lens just below the anterior capsule, and since the lens usually shows opacification at various depths as the result of penetration of the foreign body, it is necessary so to direct the beam of light of the slit lamp that it does not penetrate to the more posterior opacities of the lens. The focal beam of light must be brought in from an extremely temporal position, with the pupil widely dilated. In this manner, the ray of light will strike over the anterior capsule only and will not pass into the other layers of the lens. If the rays of light strike more deeply into the lens, the opacities existing in the depths of the lens will be rendered visible, and the great play of light on these opacifications will be so intense that the more anterior, and less opaque, "sunflower" will become invisible. The character of the "sunflower" is such that when there is an opacity posterior to it, its morphologic features cannot be recognized either by focal illumination or by retroillumination.

When the beam from the slit lamp is brought in from the extreme temporal side and the "sunflower" is viewed through the microscope, approximately the nasal half can be seen. This is due to the fact that the dilated and thickened iris acts as a barrier so that the light does not reach the temporal half of the anterior capsule. If the angle of incidence is made more acute, more of the temporal portion of the anterior capsule may be seen; but as the light passes from the temporal to the nasal side, the thin zone of the "sunflower" is by-passed, the nasal portion of the "sunflower" becoming visible.

REPORT OF A CASE

An Army private aged 19 stated that in December 1942, while he was still a civilian, a 22 mm. rifle with which he was practicing accidentally "backfired" and exploded directly into his eye. It is assumed that the patient failed to close his eye at the moment of "backfire," for the injury was direct to the eyeball. Several small copper particles were removed by the nurse, for the family physician was not available at the time of injury. However, when the patient returned the next day, his physician recommended that he use an ointment and drops several times daily. One week later, because the eye had failed to respond to this treatment, the patient consulted an ophthalmologist, who removed many residual fragments from the right cornea. The pupil was dilated, and the patient was told that deeply embedded foreign bodies were still present in the cornea and that they had better be left alone. No mention was made of the penetration of any particles through the cornea or of the presence of any intraocular foreign elements. Three weeks later, when the patient returned to the ophthalmologist, vision was as good in the right eye as it was in the left eye.

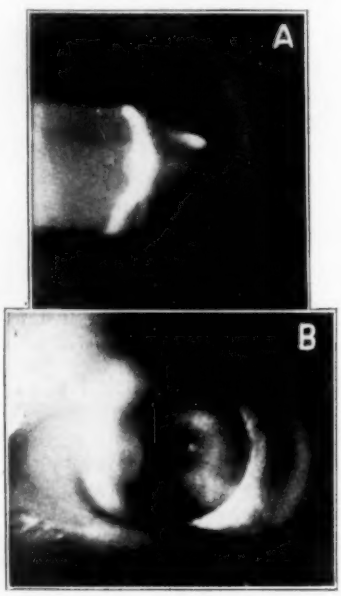
About one week later, while playing basketball, the patient was jabbed in the right eye by a finger of one of the players. The eye became painful and blurred soon after this incident, but since the patient had been told not to return to his physician for three weeks, he followed this direction literally. On the occasion of his next visit, the patient was told that the second injury was much more serious than the first and that a "film" had now formed on the eye. The patient returned to this physician several times and eventually was advised that the eye would improve. On May 22, 1943 he was inducted into the Army, with a visual record of 20/40

1. Cordes, F. C., and Harrington, D. O.: Bilateral Absorption of Intraocular Copper with Chalcosis in One Eye, Am. J. Ophth. **18**:348, 1935. Coppez, H.: Sur la chalcose oculaire, Arch. d'opht. **45**:609, 1928. Ertl, F.: Fremdkörper (Kupfersplitter) im Glaskörper, Centralbl. f. prakt. Augenh. **31**: 322, 1907. Weiss, W.: Zur Entstehung der Verkupferung des Auges durch intraokuläre Kupfersplitter, Arch. f. Ophth. **117**:114, 1926. Sala, G.: Sulla calcosi oculare con particolare riguardo all'aspetto biomicroscopico della cataratta de rame, Boll. d'ocul. **13**:1047, 1934.

for his right eye. A letter subsequently received from the aforementioned ophthalmologist stated that "several particles of powder and corneal scars were of negligible importance. The serious involvement was plastic iritis, secondary to injury from a finger nail."

The patient was admitted to the ophthalmic service of the station hospital, where several interesting observations were made.

1. Vision on admission was 20/70 in the right eye and 20/20 in the left eye. There was no apparent involvement of the adnexia, and the conjunctiva of neither eye showed injection. Muscle balance was normal in all the cardinal directions of gaze, with the near point of convergence 95 mm. On the temporal side of the bulbar conjunctiva of the right eye two small foreign bodies were embedded just beneath the conjunctiva. The cornea showed several areas of scar formation and some areas in which metallic foreign bodies were still deeply embedded in the cornea. One small foreign body, extending horizontally across the cornea for a distance of 2 mm. on a level with the lower border of the pupil, had the distinctive color of copper. Just above this was a small, opaque corneal scar, located at about 9 o'clock, but about 3 mm. within the limbus. This was the most important corneal disturbance, being the opacity produced by the penetrating foreign body. When the narrow beam of the slit lamp was passed through this opacity, the penetrating nature of the corneal scar could be determined. At the endothelial terminus of the corneal scar a small indentation could be seen, breaking up the parallelism of the corneal beam.



A, "glass tubings," visible just behind the posterior bulge; *B*, nasal half of the "sunflower," seen with the slit lamp at a wide angle of incidence.

2. There was no cellular deposit in the anterior chamber and no evidence of recent or old iritis. The zone of specular reflection, playing off the endothelial surface of the cornea, failed to show any "crater formation," and no disturbance was noticed in the endothelial "mosaic" except in the region of penetration of the corneal injury described in the preceding paragraph.

Following an angle of about 25 degrees with the visual axis, a rhomboid defect was seen in the iris at about the region of the "collarette," at 9 o'clock. The color of the iris in the injured eye was similar to that of the iris of the left eye.

When the pupil was dilated and the same angle followed (which represented the trajectory of the intraocular missile), a dense, irregularly circular opacity was seen on the anterior capsule. This lesion was superficial, grayish white and imbricated. This opacity did not extend below the surface of the lens.

3. With the narrow beam of the slit lamp passing through the various layers of the lens, many interesting changes were noted. Just below the reduplication line of the anterior capsule was a row of dotlike, bluish green opacities, in parallel arrangement, filling the entire section of the corneal beam and extending from the upper to the lower border of the iris. As the thickness of the corneal beam was increased, these minute dots could be seen to be part of the "sunflower."

When the "sunflower" was viewed with the diffuse beam of light from the extreme temporal side, the exact size of the lesion could be determined. It consisted of an inner circle, 2.5 mm. in diameter, containing an almost optically empty center. Radiating from this ring were several narrow "petals," of variable length and microscopically made up of small, round, isolated dots, so closely packed together that the entire structure seemed homogeneous. The "sunflower" was aquamarine under focal illumination. There were approximately forty petals in the structure of the lesions.

4. In the posterior portion of the lens, another opacity was visible, which by reflected light seemed to have the appearance of a "bomber flying directly overhead, with most of the tail torn away." With the narrow beam of the slit lamp, the posterior portion of the lens could be seen to bulge backward in a characteristic manner through the space represented by the outline of the framework of the wing of the plane, the bulge producing a strangely unique traumatic lenticonus posterior. The aforementioned "framework of the wing" was seen to coil on itself and produce a reduplication line, the opacity calling to mind glass tubings.

The entire opacity in the posterior section of the lens was much more dense than that in the region of the "sunflower," and whenever a ray of light was so directed that it struck the posterior part of the lens, the anterior part of the "sunflower" was rendered invisible. This posterior opacity was thickest in the central (polar) region and when viewed in the zone of specular reflection had a polychromatic luster. The reduplication lines on the posterior capsule extended across the lens, from the nasal to the temporal side.

From the posterior polar region of the opacity a long, thin, brownish line extended into the inner lenticular structure to a region just back of the posterior Y suture.

The anterior portion of the vitreous showed many indications of degeneration. The fibrous network was dense, and the interfibrillar spaces were optically empty. The latter change was present for only a small distance posterior to the lens. The adjacent region was more dense than normal, and the vitreous had a milky appearance, with more than the normal amount of horizontal interlacing fibers. In this area there was some play of color, the horizontal small lines appearing reddish with a noticeable interspersion of blue. Intermixed with these fibers were several irregular,

vertically running, rootlike fibers, free of branching structures.

Von der Heydt,[2] Jess [3] and Vogt [4] showed that the copper particles lie in the region of the epithelial cell layer, with deposition in the protoplasm and with the nucleus of the cell remaining free of the copper substance. Vogt's histologic studies showed that the deposition of copper is mainly in the epithelium of the lens. Jess expressed the belief that the radiate appearance of the "sunflower" is related to the direction and morphologic character of the radial folds of the iris, the copper particles being deposited in the so-called depressions of the radial structure of the iris.

Purtscher,[5] in 1918, pointed out that the "sunflower" is diagnostic of retained copper. Zur Nedden [6] described a case of chalcosis lentis in which the characteristic copper substance disappeared from the lens several years after removal of the intraocular foreign body. The onset of appearance of the characteristic changes of chalcosis depends on the amount of copper introduced, the location of the foreign body and the rate of decomposition of the metal.[7] The final color and morphologic features of the "sunflower" will depend on the amount of copper carbonate which is deposited.

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3. Jess, A.: Der histologische Bild der Kupfertrübung der Linse, Klin. Monatsbl. f. Augenh. **68**: 432, 1922; Verkupferung der Zonulafasern und der teilweise abgelösten Zonulalamelle bei luxiertem Kupferstar, ibid. **76**:465, 1926; Das Verschwinden der Verkupferungserscheinungen des Auges, Centralbl. f. prakt. Augenh. **53**:172, 1929.

4. Vogt, A.: Atlas of the Slitlamp-Microscopy of the Living Eye, translated by R. von der Heydt, Berlin, Julius Springer, 1921, vol. 2; Chalcosis von Linse und Glaskörper, Klin. Monatsbl. f. Augenh. **67**:560, 1921.

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6. zur Nedden: Mitteilungen über ein eigenartiges Verhalten von Kupferstückchen im menschlichen Auge, Klin. Monatsbl. f. Augenh. **41**:484, 1903.

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Clinical Notes

RECONSTRUCTION OF ABLATED LOWER LID

HAROLD F. WHALMAN, M.D.
Clinical Professor of Ophthalmology, College of Medical Evangelists
LOS ANGELES

The axiom "Necessity is the mother of invention" finds frequent fulfilment in the sphere of surgery, particularly, perhaps, in the field of reconstructive surgery, where conditions are constantly at variance and ingenuity takes precedence over established procedures in meeting the requirement of each case. Hence the report of a newly devised procedure in no way disparages the value of previously reported methods; and if it solves a particular problem, it would seem odious to review, discuss and rationalize all other operations in order to justify the usefulness of one. The method I am about to describe arose out of the necessity for extensive ablation of the right lower lid in a case of basal

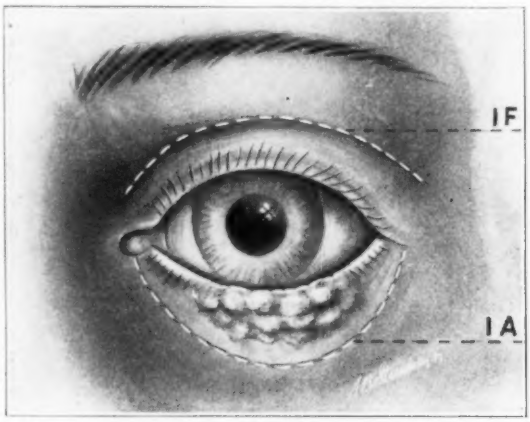

Fig. 1.—Formation of a cutaneous flap from the upper lid in the reconstruction of an ablated lower lid. *IF* indicates incision for flap, and *IA*, incision for ablation.

cell carcinoma which had grown in both directions laterally from the middlle of the lower lid and had invaded the tarsal cartilage. The growth was invasive and did not subside with moderate irradiation, and since a larger dose would have resulted in destruction of the lid, excision, with a safe margin, seemed advisable.

The lower lid was therefore excised by sharp dissection, the incision beginning at the external canthus and taking a curvilinear course through the skin down along the inferior orbital margin to about 3 mm. to the nasal side of the punctum. The tarsal plate and conjunctiva of the lid were then included in the excision, scissors being used to complete the ablation. The lower canaliculus was split for drainage.

The first step after ablation was to prepare a broad-pedicled cutaneous flap from the upper lid in the following manner: A curvilinear incision was made in the skin, in a natural crease about 5 mm. distal to the upper margin of the lid, with inclusion of the orbicularis muscle. The skin was then carefully undermined just

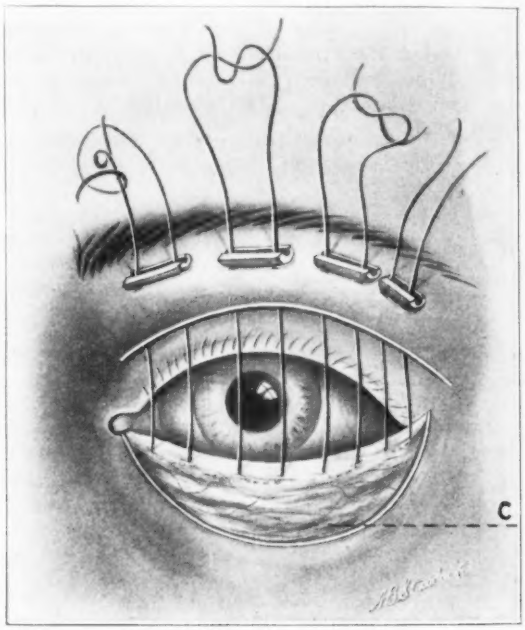

Fig. 2.—Placing of the sutures in the conjunctival flap (from lower lid) and the cutaneous flap from the upper lid. *C* indicates the conjunctiva undermined.

beneath the orbicularis muscle for a distance of 15 mm. to make a skin–orbicularis muscle flap (fig. 1).

In the second step of the operation the conjunctiva of the lower cul-de-sac was undermined down to the fornix. (There is considerable conjunctivitis in the fornix even after removal of the lid; furthermore, it can be stretched considerably after it is undermined.) Into the conjunctival flap four double-armed black silk sutures were inserted from the conjunctival side, and each, in turn was then inserted through the orbicularis muscle–skin flap, coming out in

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the skin (fig. 2). These sutures were tied over small pieces of rubber on the cutaneous side, so that the skin–orbicularis muscle flap had a conjunctival lining; of course, the eyeball was then obscured. The free margin of the flap was then united to the skin–muscle edge of the incision made through the lower lid by means of closely placed interrupted black silk sutures (fig. 3).

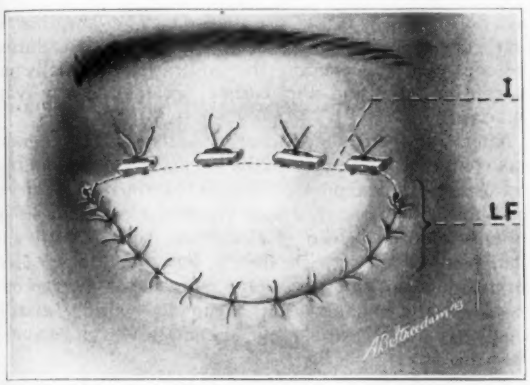

Fig. 3.—Cutaneous flap from the upper lid in place. *I* indicates final incision to free graft, and *LF*, lid flap, lined with conjunctiva (eye obscured).

Union was allowed to take place for ten days; then all sutures were removed, and the flap from the upper lid was severed from its base. The margins of the wound in the upper lid were trimmed and united, and the edges of the skin and conjunctiva of the new lower lid were sutured together with interrupted stitches, so that a cleanly united upper lid was left and a cutaneous flap replaced the ablated skin of the lower lid (fig. 4).

The new lower lid was actually larger than needed, but the excess was not trimmed to allow for shrinkage.

Two months later little shrinkage had taken place; so the lower lid was reduced to better size by excising some of it at its margin.

The lower canaliculus, which had been split, remained patent; and because the lower lid of the fellow eye had imperceptible eyelashes, no attempt was made to implant hair-bearing skin for lashes. It is my opinion that lashes on the lower lid are usually so inconspicuous that the procedure is not always necessary for good appearance.

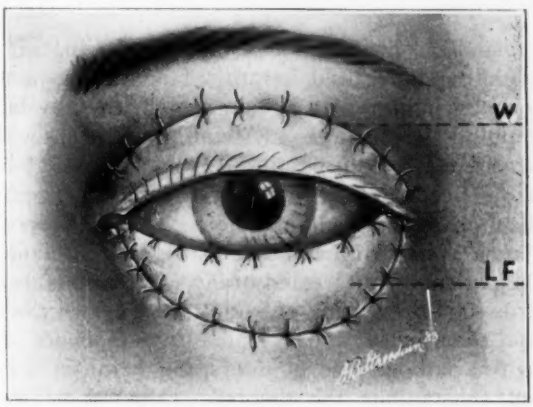

Fig. 4.—Final step in reconstruction of the ablated lower lid. *W* indicates wound in upper lid united, and *LF*, lid flap excised from the base, ten days after union.

Exceedingly good function and appearance have resulted from the operation described. The lid is fully reconstructed except for the cartilage, which does not seem to be important in the lower lid. The finished work is smooth and even with no perceptible scars, as union takes place in natural folds of the upper, as well as the lower, lids.

727 West Seventh Street.

UNSUCCESSFUL TREATMENT OF SYPHILITIC INTERSTITIAL KERATITIS WITH SULFANILAMIDE

Roy O. Scholz, M.D.
BALTIMORE

Sandler [1] published a case in which congenital syphilitic interstitial keratitis failed to respond to antisyphilitic and foreign protein therapy but rapidly cleared after systemic treatment with azosulfamide (disodium 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′, 6′-disulfonate) and sulfanilamide. After this report Arena [2] reported 7 additional such cases in which the patients were treated orally with sulfonamide drugs. The clearing of symptoms was described as "remarkable." He stated that evidence as to the pathogenesis of interstitial keratitis does not substantiate the theoretic value of sulfonamide compounds but he felt that his empiric results justified the use of these drugs in conjunction with antisyphilitic therapy.

Four patients with congenital syphilitic interstitial keratitis were treated in the Johns Hopkins Hospital with ordinary antisyphilitic therapy plus sulfanilamide in larger doses than was used by Arena, and in none of these was there any significant improvement which might be attributed to this therapy. One of these patients was also given a second course of treatment with sulfanilamide plus riboflavin, without effect. In all 4 patients the usual chronic course of the disease followed,[3] inflammation gradually subsiding as corneal vascularization became complete.

REPORT OF CASES

Case 1.—D. S., a 9 year old white girl, was admitted ten days after the onset of interstitial keratitis. The serologic test for syphilis gave a positive result, and other stigmas of congenital syphilis were present. She received one injection of mapharsen and doses of sulfanilamide (1 Gm. three times a day) for eleven days, an average level of 4 mg. per hundred cubic centimeters of blood being maintained. The keratitis at first consisted of central, deep infiltration, but during the course of this therapy the infiltration gradually increased in size and the cornea became heavily vascularized.

After the patient's discharge antisyphilitic therapy was continued, and two months later she was readmitted because of extreme photophobia and lacrimation. During this hospitalization she was given the same dose of sulfanilamide as before (1 Gm. three times daily) and riboflavin (5 mg. twice daily) for nine days. The keratitis did not improve. One month after her second discharge the inflammation began to subside, and it completely disappeared approximately seven months after the onset.

Case 2.—L. M. S., a 21 year old Negro woman, was found to have a positive serologic reaction for syphilis in a routine examination. She had had poor vision since the age of 11 but had had no active symptoms until one month before admission, when her left eye became sore. Six months prior to admission she had had a generalized cutaneous eruption. Her condition was diagnosed as congenital syphilis and syphilitic keratitis of the left eye.

She had received injections of mapharsen and was receiving her first doses of bismuth when she was admitted for systemic treatment with sulfanilamide. The right eye was essentially normal, and the left eye showed the typical picture of syphilitic interstitial keratitis. The vessels extended about 4 mm. from the limbus, and there was a mild complicating anterior uveitis. She was given 3 Gm. of sulfanilamide on admission and 1 Gm. every four hours thereafter, a level of 7.5 mg. per hundred cubic centimeters of blood being reached. During the eleven days of this treatment the vessels regressed slightly and the eye became somewhat clearer. She was discharged, showed slight improvement for three months and then had a recurrence in the right eye, which remained abnormal for an additional six months.

Case 3.—F. M. J., a white youth of 16, was admitted because of a positive serologic reaction for syphilis and interstitial keratitis. The patient's mother also gave a positive reaction to a serologic test for syphilis. At the age of 10 he had had a penile lesion. His left eye was sore for six weeks prior to admission. His right eye showed marked lacrimation, blepharospasm and conjunctival injection but no corneal lesion. The left eye showed a typical picture of interstitial keratitis with photophobia, lacrimation and blepharospasm. The cornea was gray and fairly opaque, with small vessels progressing from the limbus almost to the center. He was given sulfanilamide, 1 Gm. every four hours for eight days, a level of 5 mg. per hundred cubic centimeters of blood being maintained. No great difference was noted either in the clearness of the cornea or in

From the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital.

Mary S. Goodwin, M.D., of the Syphilis Division of the Medical Clinics, cooperated in the study of these cases.

1. Sandler, I. L.: Sulfanilamide Treatment of Syphilitic Keratitis: Preliminary Report, Arch. Dermat. & Syph. **39**:528 (March) 1939.

2. Arena, J. M.: Use of Sulfonamides in Treatment of Syphilitic Keratitis, J. Pediat. **20**:421 (April) 1942.

3. Woods, A. C.: Syphilis of the Eye, Am. J. Syph., Gonor. & Ven. Dis. **27**:133 (March) 1943.

the reduction of the infiltrating vessels when the patient was discharged or when seen three weeks later.

CASE 4.—M. H., a Negro girl aged 11 years, had had her first attack of interstitial keratitis at the age of 5. Both of her parents gave a history of having received antisyphilitic treatment. She responded well at that time to syphilitic therapy and continued to take routine treatments with bismuth and arsphenamine. Eight weeks before admission she began to have difficulty with walking, and for six weeks before admission she had increasing deafness and complained of tinnitus. Four weeks before admission the left eye had a recurrence of interstitial keratitis.

The child was completely deaf, and examination was difficult. The right eye presented the picture of old healed interstitial keratitis. The left eye showed extreme photophobia and lacrimation. There were a number of infiltrations scattered over the cornea and numerous layers of deep dilated vessels, which covered the whole cornea. She was given 1 Gm. of sulfanilamide every four hours, a level of 14 mg. per hundred cubic centimeters of blood being maintained. During her three weeks' hospitalization the photophobia and lacrimation almost completely disappeared, and the congestion subsided. The vascular picture and the infiltrations remained the same. Vision could not be tested because of the complete deafness of the child. She was seen in the dispensary one month later, when further improvement was noted. This is the only patient in whom a suggestion of improvement was found, and that improvement took place only after the vessels had coalesced.

SUMMARY AND CONCLUSIONS

The addition of large doses of sulfanilamide to the usual antisyphilitic therapy in the treatment of 4 patients with congenital syphilitic interstitial keratitis did not prove of any benefit. Healing of the cornea took place, as usual, only after vascularization was complete. I do not believe sulfanilamide therapy is indicated in the treatment of this disease.

Ophthalmologic Reviews

EDITED BY DR. FRANCIS HEED ADLER

MODERN TRENDS IN OCULAR THERAPEUTICS

ROY O. RISER, M.D.
PARK RIDGE, ILL.

This summary of certain trends in ocular therapeutics was made at the request of the Committee on Scientific Exhibits of the American Academy of Ophthalmology and Otolaryngology. The material has been taken from rather recent literature and is not a complete review. Complete bibliographies will be found in some of the latest articles.

THE EYELIDS

Lesions of the dermal surface of the eyelids and of the lid margins may be associated with infections of the conjunctiva or may result from the use of medicaments, such as physostigmine or atropine, for deeper diseases of the eye. Dermatologic diseases or systemic disturbances may also affect the lids.

Some lesions of the lids are best treated by specific medication directed at the bacterial cause, such as the use of sulfathiazole ointment and staphylococcus toxoid for staphylococcic blepharitis. Vitamin therapy or regulation of diet is of aid in the therapy of other conditions. Allergic reactions involving the lids and angioneurotic edema may require special tests and still other forms of treatment.

The seborrhea of puberty requires different treatment than does stagnation of the sebaceous glands of senility. The relative value of oily medicaments, such as cod liver oil, and of fat-dissolving solutions, such as purified benzine (to be used with caution), for cleansing dry and crusted lids must also be given careful consideration, as stressed by Linksz.[1] He emphasized the necessity of cleansing the lids but warned that soap, water and hot packs may perpetuate certain conditions.

Many disturbances of the lids are easily made worse by overmedication. The best therapy is often the simplest and mildest dermatologic preparation that makes the patient comfortable while the cause is being investigated and eliminated. Linksz described the pharmacologic action of the following types of such preparations.

Powders.—Powders such as talc and zinc oxide may be chiefly protective, or magnesium carbonate may be used for its adsorbing and mild antiphlogistic effect. Oozing or weeping surfaces must not be treated with powders.

Lotions.—Powder in water, with or without glycerin, is also used for dry lesions.

Calamine Lotion N. F.: This preparation constitutes the standard treatment for simple dermatitis.

Disinfectant Lotion (Lutz): Use of such a lotion is advisable in treatment of eczematous impetigo.

	Gm. or Cc.
Resorcinol	0.2
Ichthammol	0.5
Zinc oxide	
Talc	āā 1.5
Dilute alcohol	
Distilled water	āā ad 10.0

The danger of resorcinol dermatitis should be borne in mind.

Cold Cream.—"Cold" cream, or "refrigerating cream" (rose water ointment U. S. P.), is especially good for dry skin. The essential oils in cold cream may be irritating.

A substitute for cold cream, to be used for irritated lids, may be made up as follows:

	Gm. or Cc.
Wool fat	
Boric acid, 2 per cent solution	āā 10
White petrolatum	5

An astringent and mild disinfectant (Seibert) may be desired.

	Gm. or Cc.
Solution of aluminum acetate	2
Distilled water	5
Wool fat	
White petrolatum	āā 10

Pastes.—Pastes are mixtures of powder and grease and are useful in treatment of broken or weeping surfaces or of surfaces macerated by persistent epiphora. The following preparations are mentioned:

Lassar's Zinc Oil (for maceration and for fissures):

Zinc oxide	6 Gm.
Olive oil	4 Gm.

1. Linksz, A.: Applied Pharmacology of the Skin in the Ophthalmologist's Everyday Practice, Arch. Ophth. **28**:959 (Dec.) 1942.

Ichthammol (0.5 Gm.) may be added to increase the antiseptic properties.

Lassar's Zinc Paste (for dermatitis, such as that due to atropine or hair dye):

Zinc oxide	
Talc	
Wool fat	
White petrolatum	āā 10 Gm.

Atkinson's Paste (for eczema or herpes zoster of the lids):

Bismuth subnitrate	2.5 Gm.
Wool fat	
Zinc oxide ointment U. S. P.	āā 15.5 Gm.

"Refrigerating Pastes."—These preparations can be applied to most irritated skins. Linksz stated that if he is in doubt as to the diagnosis of a lesion of the lids or as to the best treatment he usually resorts to a refrigerating paste, which can do little harm. Linksz mentioned the following preparations.

Neisser's Paste (for chronic inflammation of the lid margins):

Zinc oxide	
Bismuth subnitrate	āā 0.5 Gm.
Rose water ointment	
Cerate U. S. P.	āā 10.0 Gm.

Substitute for Neisser's Paste (for patients sensitive to oils in rose water ointment U. S. P.):

	Gm. or Cc.
Magnesium carbonate	5
2 per cent solution of boric acid	
White petrolatum	āā 10

Stein's Paste (for fissures of lid margins):

	Gm.
Ichthammol	0.1
Zinc oxide	
Bismuth subnitrate	āā 1.0
Rose water ointment U. S. P.	10.0

Slight oozing or weeping is not a contraindication.

Ointments.—An ointment consists of an active drug in a viscous base. A variety of bases, used alone or in combination, are available.

1. Petrolatum as a base is used chiefly for chronic lesions when deeper penetration of the active drug is desired. Water-soluble drugs cannot be applied in petrolatum, but it may serve as a base for other drugs. Peter's ichthyol zinc ointment, for the treatment of rosacea of the lids, is such a preparation.

	Gm.
Ichthammol	0.15
Zinc oxide	0.50
White petrolatum	15.00

2. Wool fat is usually combined with petrolatum because it forms an emulsion with water and watery solutions of active drugs. Such a preparation is bismuth tetralnomopyrocatechin (noviform), used for chronic blepharitis in 10 per cent concentration in an ophthalmic ointment base made of wool fat, water and petrolatum.[2]

3. A base made of cholesterol derivatives and petrolatum is also miscible with drugs in aqueous solutions. This has been used for seborrhea of the lid borders by Gifford.[3]

	Gm.
Naphthalan	2.0
Ichthammol	0.6
Zinc oxide	
Starch	āā 8.0
Oxycholesterol-petrolatum base	q.s. 30.0

4. Wetting Agents: Commercial detergents of low surface tension, although still in the semi-experimental stage for ophthalmic medication will undoubtedly be the next addition to ophthalmic ointment bases. For further details the reader is referred to the last paragraph of the section on "Better Eye Drops."

GLAUCOMA

The following therapeutic agents are available.

Acute Glaucoma.—1. Physostigmine salicylate (or sulfate), in 0.5 to 2 per cent solution. Five drops is instilled at one minute intervals, and the instillations may be repeated in one hour and, again, in three hours, as indicated. The solution may be combined with a 2 per cent solution of pilocarpine nitrate, if desired.

2. Morphine sulfate, given by hypodermic injection, for pain. Administration may aid in induction of miosis.

3. Magnesium sulfate, as a purgative, for reduction of fluids.

4. Hypertonic solutions given intravenously, for reduction of fluids.

(*a*) Sodium chloride: Fifty cubic centimeters of a 30 per cent solution (or 150 cc. of a 10 per cent solution). It acts the most rapidly of the three solutions indicated, being effective in less than five to eight hours.

(*b*) Dextrose: One hundred cubic centimeters of a 50 per cent solution. The effect is slower and more prolonged than that of the hypertonic solution of sodium chloride.

2. The ointment base is made by the Manhattan Eye Salve Company.

3. Gifford, S. R.: Hand-Book of Ocular Therapeutics, ed. 3, Philadelphia, Lea & Febiger, 1942, chap. 6.

4. Manufactured by Abbott Laboratories, North Chicago, Ill.

(*c*) Sorbitol: One hundred cubic centimeters of a 50 per cent solution. The effect is greatest twelve to twenty-four hours after injection.

5. Glycerin: Corneal edema may be cleared in thirty seconds by instillation of drops of full strength glycerin. The clearing lasts for several minutes and, according to Cogan,[5] does no harm.

6. Alternatives for physostigmine. Other miotics for treatment of acute glaucoma are:

(*a*) Neostigmine bromide, in 5 per cent solution, usually used with a 20 per cent solution of mecholyl chloride. Repeated instillations may be made, with or without retrobulbar injection of 10 to 20 mg. of mecholyl chloride. This injection may have dangerous effects; the antidote is atropine. The aforementioned combination is more advisable for use in the early stages of acute glaucoma.

(*b*) Furfuryltrimethylammonium iodide (furmethide), in 10 per cent solution. One drop is instilled every fifteen minutes for two hours, and then every three hours until tension becomes normal or operation is performed. This drug is more advisable for use in the later stages of acute glaucoma.

(*c*) Histamine. This substance is the most powerful miotic available, but is not advisable for use because of the severe reaction it induces.

7. Surgical intervention. Operation must not be delayed if tension remains at an unsatisfactory level for more than a few hours.

Chronic Glaucoma.—The following substances are available for therapy.

1. Pilocarpine nitrate, in a 1 to 2 per cent solution two or six times daily.

2. Physostigmine salicylate (or sulfate), in 0.25 to 0.5 per cent solution, alone or to increase the action of pilocarpine. This substance often causes sensitivity when used over long periods of treatment in chronic glaucoma.

3. Neostigmine bromide, in 3 per cent solution. This substance may be used alone, as a substitute for pilocarpine, but is usually combined with mecholyl chloride, 10 per cent, to give a synergistic effect. Mecholyl is not used alone.

4. Carbaminoylcholine chloride, in 0.75 to 1.5 per cent solution. It is much more effective in a 1:3,000 solution of zephiran chloride, giving results equal to those with a 2 per cent solution of pilocarpine nitrate or a mixture of 2 per cent pilocarpine nitrate and 0.5 per cent physostigmine salicylate (or sulfate). Clarke[6] claimed that other miotics are more effective after a period of rest induced with carbaminoylcholine.

5. Furfuryltrimethylammonium iodide, in 10 per cent solution. This substance is slightly more effective than a 2 per cent solution of pilocarpine nitrate for chronic glaucoma but, like the other new miotics, does not replace it.[7] It is an adjuvant in the medical treatment, for if one miotic is no longer tolerated another may still keep the tension lowered.

6. Epinephrine and its derivatives. These substances, while causing mydriasis, lower the intraocular tension. Miotics are to be used before and after administration of these drugs, which is not advisable in treatment of glaucoma other than the simple chronic type.

(*a*) Epinephrine hydrochloride. Four minims (0.25 cc.) of a 1:1,000 solution is injected subconjunctivally, or 5 minims (0.31 cc.) of a 1:1,000 solution on a cotton pledget is inserted in the upper cul-de-sac for four minutes (Gradle's method).

(*b*) Synthetic dextrorotary epinephrine 2 per cent solution.

(*c*) Epinephrine bitartrate in 2 per cent solution (J. Green).

(*d*) Neo-synephrine hydrochloride either in 10 per cent emulsion or in 10 per cent solution in the new low surface tension vehicle (Frederick Stearns & Company).

The treatment of glaucoma is discussed by Gifford[8] in greater detail.

NOTE OF CAUTION.—The action of these agents is increased by promotion of the general health, elimination of other disease when possible and abstinence from tobacco, coffee and table salt. The visual fields should be watched, and tonometric records should be kept. Surgical treatment may be indicated.

INDICATIONS FOR SULFONAMIDE COMPOUNDS IN OPHTHALMIC PRACTICE

Oral Administration.—To obtain standard concentrations in the blood of 5 to 10 mg. per hundred cubic centimeters, administration of at least 0.5 Gm. (7.7 grains) every two hours for adults is advised. If several sulfonamide compounds have been employed or recommended for

5. Cogan, D. G.: Clearing of Edematous Corneas by Glycerine, Am. J. Ophth. **26**:551 (May) 1943.

6. Clarke, S. T.: Use of Doryl in Treatment of Glaucoma, Am. J. Ophth. **25**:309 (March) 1942; Mecholyl and Prostigmine in Treatment of Glaucoma, ibid. **22**:249 (March) 1939.

7. Uhler, E. M.: Use of Furmethide in Comparison with Other Miotics for Treatment of Glaucoma, Am. J. Ophth. **26**:710 (July) 1943.

8. Gifford,[8] chap. 11.

a certain disease, with apparently equally good results, sulfadiazine is indicated because it is least toxic. Foreign protein, when indicated, may be given with full doses of the chosen sulfonamide compound.

Local Use.—The substances are given as powders, solutions, emulsions and ointments and by iontophoresis, and now incorporation of the less permeable sulfonamide compounds in "wetting agents," which Bellows [9] has tried experimentally, promises even greater local use of medicaments at present given internally, with due consideration of possible toxicity. Insufflation of powders every three to four hours is useful if the patient is hospitalized.

A discussion of the chemotherapy of infections of the eye by Thygeson [10] is summarized in the following tabulation.

Disease	Organism	Oral or Local Use	Sulfonamide Compound Indicated
Trachoma	Virus	Oral	Sulfanilamide still advisable
Inclusion conjunctivitis	Virus	Local (oral as indicated)	Sulfathiazole
Lymphogranuloma venereum	Virus	Oral	Sulfadiazine
Gonorrheal ophthalmia	Gonococcus	Oral (powder applied locally is optional)	Sulfadiazine
Erysipelas (lids)	Streptococcus B haemolyticus	Oral	Sulfanilamide still advisable
Impetigo		Local	
Dacryocystitis (acute)		Oral	
Conjunctivitis		Local	
Corneal ulcer		Both	
Panophthalmitis		Oral	
Blepharitis	Staphylococcus	Local (and staphylococcus toxoid)	Sulfathiazole
Conjunctivitis		Local	Sulfathiazole
Marginal corneal ulcers		Local (powder)	Sulfadiazine
Impetigo		Local	Sulfathiazole
Recurrent hordeolum		Local	Sulfathiazole
Corneal ulcer	Pneumococcus	Both (powder)	Sulfapyridine
Conjunctivitis	Pneumococcus	Local	Sulfathiazole
Corneal ulcer	Bacillus Friedländer or Bacillus pyocyaneus	Both (powder or by iontophoresis)	Sulfadiazine
Conjunctivitis	Several	Local (other medications are not to be discarded)	Sulfathiazole
Penetrating injury	Several	Oral (local, optional)	Sulfadiazine
Sympathetic ophthalmia	?	Oral	Sulfanilamide (Not a cure)
Endophthalmitis	Meningococcus and others	Oral (and by iontophoresis [?]) (penicillin probably of more avail for such metastatic lesions)	Sulfanilamide Sulfadiazine?
Uveitis	Several	Oral, if at all	Sulfadiazine(?)

A number of reports, personal experiences, and the like, indicate that the sulfonamide compounds are disappointing in treatment of uveitis. They may be tried with foreign protein therapy; administration of salicylates; specific therapy, such as use of tuberculin or antisyphilitic drugs, and removal of foci of infection.

BETTER EYE DROPS

The following factors are to be considered in the evaluation of eye drops: [11]

1. *Tonicity.*—Solutions isotonic with tears are generally least irritating. Tears are isotonic with a 1.4 per cent solution of sodium chloride.

2. *Hydrogen Ion Concentration.*—The acidity or the alkalinity is a common, and more important, cause of irritation from eye drops. This is measured in terms of the p_H, which indicates the hydrogen ion concentration. The p_H also influences the solubility and activity of the drug. Pure water has a p_H of 7; acids have a p_H of less than 7, and alkalis, a p_H of more than 7. The more nearly the p_H of the eye drops approaches the average p_H of tears (7.5), the more com-

9. Bellows, J.: Chemotherapy in Ophthalmology, Tr. Am. Acad. Ophth. **47**:19 (Sept.-Oct.) 1942; Arch. Ophth. **29**:888 (June) 1943.

10. Thygeson, P.: Sulfonamide Compounds in Treatment of Ocular Infections, Arch. Ophth **29**:1000 (June) 1943.

11. Various problems associated with ophthalmic solutions have been discussed by the following authors: Elvin, N. C.: The p_H and Tonicity of Ophthalmic Solutions, Arch. Ophth. **29**:273 (Feb.) 1943. Gifford,[3] chap. 3. Gifford, S. R.; Puntenney, I., and Bellows,

(Footnote continued on next page)

fortable they are; but the drugs vary with respect to such factors as chemical composition, solubility and optimum rate of absorption.

3. *Buffer Solutions.*—Such solutions resist changes in p_H by diluents, such as the lacrimal secretions. An almost constant p_H can be maintained by the correct selection of the type and proportion of acid or alkaline constituents. Buffered eye drops are less irritating, and the drugs they contain are more readily absorbed.

The recommended hydrogen ion concentrations, in terms of the p_H of various forms of buffered eye drops, is tabulated as follows:

Ophthalmic Solution

p_H	
5.0	Butacine sulfate; phenacaine hydrochloride (the solution must be warmed)
5.9	Eucatropine hydrochloride, in a special buffer formula
6.0	Zinc, cocaine, epinephrine, paredrine hydrobromide ophthalmic and 2 per cent boric acid
6.7	Tetracaine hydrochloride in isotonic solution of sodium chloride (do not buffer)
6.75	Special buffered, isotonic, preserved solution of zinc sulfate, recommended by Arrigoni and associates [12]
7.6	Homatropine, atropine, scopolamine, physostigmine, pilocarpine. Buffer solution for use alone, as a general collyrium
8.4	A buffer solution alone, for the secretions of vernal catarrh
8.6 to 8.8	Buffered solutions for wearers of contact lenses [13]
9.0	Vehicle for fluorescein, requiring alkaline solution (fluorescein sodium is soluble in pure water)

Easy Method of Prescribing for Buffer Solutions: With the buffer solutions recommended by Gifford and Smith [14] and Feldman,[15] mixtures of two stock solutions are used in varying proportions to obtain the required p_H. To 30 cc. of the acid stock solution, a standard fixed amount, is added a smaller amount of alkaline stock solution to produce the p_H desired. One may specify Gifford's buffer solution or Feldman's buffer solution to the druggist in the manner shown in the following prescriptions, for practically all the pharmacies of the country receive the "Blue Book," [16] which contains the exact information they need to fill such prescriptions:

℞	Atropine sulfate	0.15 Gm.
	Gifford's buffer solution, p_H 7.6	15 cc.
℞	Zinc sulfate	0.02 Gm.
	Feldman's buffer solution p_H 6.0	15 cc.

4. *Preservatives* (for prevention of yeast or fungus growths).—The following substances are suggested:

Boric acid, 40 grains (2.6 Gm) to the ounce (31 Gm.) or in the proportion used in buffer solutions.

Chlorobutanol, 0.5 or 1 per cent. This substance will not change the p_H of buffer solutions.

Camphor or menthol, 1 grain (0.065 Gm.) to the ounce.

Benzoic acid esters are ideal preservatives but are not universally obtainable. The propyl and methyl esters [17] may be used in stock buffer solutions. They are especially good for preventing molds in damp climates.

5. *Substitutes for Tears.*—Buffer solution p_H 7.6 may be used as a collyrium. Use of gelatin in Locke's solution was advised by Rucker for deficiencies in secretion of the lacrimal gland (keratoconjunctivitis sicca).

Gelatin 0.3 Gm.; chlorobutanol, 0.15 Gm.; Locke's solution 30.0 cc.

Locke's solution consists of sodium chloride, 0.9 Gm.; potassium chloride, 0.024 Gm.; calcium chloride, 0.042 Gm.; dextrose, 0.1 Gm.; sodium carbonate, 0.02 Gm., and distilled water, 100 cc.

6. *Surface Tension.*—The surface tension of medicated eye drops (and of ophthalmic ointments) assumes increasing importance in the light of recent reports.

Emulsions of Oil in Water: These have been tried, especially for mydriatics and cycloplegics, but have not yet found general favor.

Aqueous Vehicles of Low Surface Tension: These preparations are more practical. One developed for neo-synephrine ophthalmic solution (see section 6 *d* under "chronic glaucoma") is now offered as a general ophthalmic

J. G.: Keratoconjunctivitis Sicca, Arch. Ophth. **30**:207 (Aug.) 1943. Hosford, G. N., and Hicks, A. M.: Hydrogen Ion Concentration of Tears: Its Relation to Certain Ocular Symptoms and to Conjunctival and Corneal Lesions, ibid. **13**:14 (Jan.) 1935. Swan, K. C., and White, N. G.: Corneal Permeability: Factors Affecting Penetration of Drugs in Cornea, Am. J. Ophth. **25**:1043 (Sept.) 1942.

12. Arrigoni, L.; Fischer, L., and Tozer, G. A.: Ophthalmic Zinc Sulfate Solutions: Buffered, Isotonic and Preserved, Arch. Ophth. **26**:852 (Nov.) 1941.

13. Obrig, T.: Contact Lenses, New York, Obrig Laboratories, Inc., 1942, chap. 9.

14. Gifford, S. R., and Smith, R. D.: (*a*) Effect of Reaction on Ophthalmic Solutions, Arch. Ophth. **9**: 227 (Feb.) 1933. (*b*) Gifford, S. R.: Reaction of Buffer Solutions and of Ophthalmic Drugs, ibid. **13**:78 (Jan.) 1935.

15. Feldman, J. B.: p_H and Buffers in Relation to Ophthalmology, Arch. Ophth. **17**:797 (May) 1937.

16. American Druggist Blue Price Book, revision 15, 1943, blue section, pp. 31 and 32.

17. Available as nipagin M and nipasol M, respectively, from the Goldschmidt Corporation, New York.

vehicle.[18] It contains a detergent, or "wetting agent, benzoic acid esters as preservatives and an isotonic solution of sodium chloride. It is compatible with fourteen common ophthalmic drugs.

"Wetting Agents": These represent the latest trend toward more efficient ophthalmic drops and ointments. Substances which lower interfacial tension between the corneal epithelium and the surrounding media produce an increased penetration of the cornea by the medicament in the wetting agent. A new field for medication of the anterior segment, especially for drugs not easily absorbed by the cornea, has been shown by O'Brien and Swan[19] in their use of zephiran chloride (a wetting agent) in augmenting the action of carbaninoyl choline chloride and by Bellows and Gutmann[20] in their use of the sulfonamide compounds with commercial wetting agents, such as the aerosols, the tergitols and the duponols.

120 Main Street.

18. Obtainable from Frederick Stearns & Company, Detroit.

19. O'Brien, C. S., and Swan, K. D.: Carbaminoylcholine Chloride in Treatment of Glaucoma Simplex, Arch. Ophth. **27**:253 (Feb.) 1942.

20. Bellows, J. G., and Gutmann, M.: Application of Wetting Agents in Ophthalmology, with Particular Reference to Sulfonamide Compounds, Arch. Ophth. **30**:352 (Sept.) 1943.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Cornea and Sclera

SOLAR KERATOCONJUNCTIVITIS ASSOCIATED WITH AMBLYOPIA. C. BERENS and P. T McALPINE, Am. J. Ophth. **27:** 227 (March) 1944.

Berens and McAlpine give the following summary:

"1. Two brothers were exposed to an open carbon-arc lamp for three 15-minute periods within 48 hours. 2. Following this, vision was reduced to the perception of hand movements and the visual fields to a temporal crescent in each eye. 3. There was gradual recovery in the course of 11 days, both of visual acuity and visual fields. 4. Consideration of effects of ultraviolet radiation make it probable that the contraction of the visual fields and temporary amblyopia in these patients were caused by the thermal effect of the carbon-arc lamp."

W. S. REESE.

Experimental Pathology

EXPERIMENTAL CORNEAL ULCERS. J. M. ROBSON, Brit. J. Ophth. **28:** 15 (Jan.) 1944.

In the course of the work on experimental ulcer and infections of the cornea of various types, particularly experimental mustard gas lesions, it was found that secondary infection played an important part in determining the severity of the lesions produced by the mustard gas (dichloroethyl sulfide). Local treatment of rabbits with chemotherapeutic agents decreased the severity of the primary reaction and the liability to the development of delayed "vascularized keratitis." These experiments suggested that chemotherapeutic agents might be of value also in the treatment of lesions of the human eye due to mustard gas, and the treatment with sodium sulfacetimide is to some extent based on this work.

In the human subject, many organisms may be concerned in the production of ulcer of the cornea. With few exceptions, however, these various organisms do not produce a characteristic clinical picture. The hypopyon type is usually due to the pneumococcus, but to some extent the etiologic agent varies with the geographic location and with the occupation of the patient. Thus, in France for example, the diplobacillus of Morax (Haemophilus duplex) and that of Petit are not infrequently concerned. In Palestine the Koch-Weeks bacillus (Haemophilus influenzae) was recovered. In the Glasgow district Staphylococcus aureus was recovered in 25 per cent of the ulcers examined.

The author describes the technic used in the experimental investigations on animals.

Pyocyaneus Ulcer.—Joy showed that the lesions were favorably influenced by the oral administration of sodium sulfapyridine. The local application of a sulfonamide compound also had a favorable effect on the course of such lesions. When a 30 per cent solution of sodium sulfacetimide was applied within one hour of inoculation, ulceration was completely prevented in more than half the treated eyes. When administered after a delay of twelve hours, its value was greatly reduced, though there was still a definite effect.

Pneumococcic Ulcer.—The best results were obtained with penicillin, though striking effects were also seen with sodium sulfacetimide. When treatment with penicillin was started within one hour after inoculation, ulceration was completely prevented in 7 of 8 eyes.

Lesions Due to Staph. Aureus.—The local application of penicillin produced striking results. Sodium sulfacetimide, though less effective, had a definite effect.

Lesions Due to Streptococcus Haemolyticus.—When treatment with penicillin (220 units per cubic centimeter) was started one hour after inoculation, the development of the lesion was almost completely inhibited.

The investigation of the treatment of tuberculous lesions is still in an early stage.

W. ZENTMAYER.

Instruments

A REPORT ON DEFECTS FOUND IN TONOMETERS EXAMINED AT THE CHECKING STATION OF THE NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS. M. J. SCHOENBERG AND A. POSNER, Am. J. Ophth. **27:** 368 (April) 1944.

Schoenberg and Posner reach the following conclusions:

"A large percentage of tonometers (Schiøtz type) contains defects of construction and of performance. These defects point to one of the two reasons for the unreliability of many tonometers (the other reason is defective technique).

"Every tonometer already in use should be checked at a reliable checking station at the earliest possible time.

"Inaccurate tonometers should not be used until the important defects are corrected or adjusted.

"It is urgent that a number of reliable checking stations be established under the direction of ophthalmologists in appropriate locations, throughout this country.

"It is important that precision mechanics be found who are able to put defective tonometers in good working order.

"Manufacturers of tonometers should be urged to have their instruments checked and certified at reliable checking stations before placing them on the market."

W. S. REESE.

Lens

THE DEVELOPMENT OF A SYSTEM OF INTRACAPSULAR CATARACT EXTRACTION. D. B. KIRBY, Am. J. Ophth. **27:** 124 (Feb.) 1944.

Kirby describes the development of a system of intracapsular cataract operations based on the condition of the capsule, cortex and zonule of the lens and the vitreous. In cases in which the zonule is resistant, after he has tried other procedures within the limits of safety, he strips off the zonule, according to a method he has devised.

W. S. REESE.

CHOLESTERINOSIS LENTIS. P. GEORGARIOU and O. WOLFE, Am. J. Ophth. **27:** 394 (April) 1944.

Georgariou and Wolfe give the following summary:

"Pathogenic explanation of cholesterinosis lentis is based upon the pathogenic theory of lipoidosis bulbi. Conditions found outside the crystalline lens produce an infiltration of the posterior cortex of the crystalline-lens capsule. This infiltration follows a secondary disturbance of the lipoid complex of the crystalline masses, producing the appearance of shining crystal of cholesterin.

"Cholesterinosis lentis is always a secondary cataract, and we should persist in detailed examinations to discover the primary causative factor."

W. S. REESE.

Neurology

INJURY TO THE OPTICO-CHIASMAL JUNCTION: A CASE REPORT. E. B. C. HUGHES, Brit. J. Ophth. **27:** 367 (Aug.) 1943.

The author reports a case of injury to the head from a head on motor crash. A man aged 31 sustained a fissured fracture running down in the left frontal bone, traversing the left frontal sinus and passing alone the floor of the left anterior fossa lateral to the optic canal and then down into the left middle fossa. The actual course of this fracture was verified in the course of two operative procedures, one for an epidural hematoma on the left side, which was removed through a craniotomy opening in the temporal region, and the second, undertaken sixteen weeks after the injury for drainage of the aerocele cavity and repair of the dural defect over the frontal sinus. After the first operation vision in the left eye was limited to ability to count fingers in the upper nasal quadrant. Ten weeks after the first operation the left eye showed a large island of vision in the upper nasal quadrant with macular vision of 1/60. Sixteen weeks after the injury the visual field of the right eye showed a large defect in the upper temporal quadrant and decrease of acuity in the lower temporal quadrant. After the second operation vision in the left eye improved to 6/36, as a result of enlargement of the island of vision to include the greater part of the macular vision. The field of the right eye lost its quadrantic defect but had a moderate-sized scotoma in the upper temporal portion. The site of the damage must have been either in the termination of the left nerve or in the anterior chiasmal angle—a lesion which corresponds to the "junction" type of defect. Such a lesion affects chiefly the fibers of the left optic nerve but also catches the knee of the crossed fibers from the lower nasal portion of the retina of the right eye. This case, while it fails to bring positive proof of the presence of this knee of fibers, nevertheless adds a little to the great weight of circumstantial evidence suggesting their presence. The fields are illustrated.

W. ZENTMAYER.

OCULAR MANIFESTATIONS OF NEUROSIS COMMONLY FOUND AMONG SOLDIERS. I. C. MICHAELSON, Brit. M. J. **2:** 538 (Oct. 30) 1943.

Many soldiers, on account of fear and environmental stress, acquire a neurosis, of which there are two main varieties, hysteria and chronic anxiety state, the form being determined by the individual personality type. These conditions may cause symptoms referable to the eyes; in fact, half the soldiers in the outpatients' room of the ophthalmic department of a military hospital in the Middle East presented functional complaints, consisting of defective day vision, defective night vision, asthenopia, headache, photophobia, spots before the eyes and epiphora. These conditions are then more fully described, with the diagnosis and treatment. Functional diplopia and defective day or night vision are referred to as hysterias, while asthenopia, headache, photophobia and spots before the eyes are defined as a chronic anxiety state with ocular manifestations. The necessity of the exclusion of organic disease in cases of such a condition is self evident.

The author concludes as follows: "It may be of some help to think of organic and functional disturbances coexisting in one of three different ways: (1) The association in one patient may be fortuitous. (2) A functional disturbance may be superimposed upon an organic one, modifying or aggravating the symptoms. (3) The psychic disturbance leading to upset of ocular function may appear to be an integral part of the organic process. This is noticed in disseminated sclerosis. It is not unusual in this disease for the 'functional' symptom to be the predominant one and to be present for a long period before the 'organic' symptoms arise."

This article is important and well worth careful study. ARNOLD KNAPP.

Ocular Muscles

OPHTHALMIC PRISMS, SOME USES IN OPHTHALMOLOGY. G. P. GUIBOR, Am. J. Ophth. **26:** 833 (Aug.) 1943.

Guibor gives the following summary:

"The action of prisms when placed before the eyes can be explained by the known physiologic phenomena of projection, reciprocal innervation, and synkinesis.

"Prisms are of value in the diagnosis and treatment of motor disturbances produced by anisometropia and neuromuscular defects. The types of prisms most commonly used are the round, the square, the prism rack, the rotary, and the toric.

"The nonsurgical treatment by prisms of divergence paralysis, concomitant convergent strabismus, and abducens paralysis is discussed, with figures to explain how prisms are applied therapeutically.

"It must be understood that prisms are prescribed only after a therapeutic test is employed with the fitovers. They should be prescribed on a neurologic and physiologic basis and not on mechanistic theory alone."

W. ZENTMAYER.

Retina and Optic Nerve

ANGIOMATOSIS RETINAE (VON HIPPEL'S DISEASE): RESULTS FOLLOWING IRRADIATION OF THREE EYES. F. C. CORDES and O. C. DICKSON, Am. J. Ophth. **26:** 454 (May) 1943.

Cordes and Dickson supply the following summary:

"Three eyes of two patients with angiomatosis retinae were irradiated. In the first patient, who had an early lesion in one eye, there was a marked improvement with retention of 0.8 vision, 3½ years after irradiation. In the second case both eyes were involved, the right eye being in a well-advanced stage, whereas the lesion in the left eye was an early one. Both eyes were given 1,800 r. The early lesion showed definite improvement in vision of 1.0 two years after irradiation. During this time the eye having the more advanced lesion became progressively worse, going on to amaurosis, complete detachment of the retina, and gliosis.

"From our experience with three eyes and from what is reported in the literature it appears that in early cases X-ray therapy does offer a convenient, safe means of treating these lesions. The advanced lesions have not responded to any type of therapy.

"Electrolysis, diathermy, radium, and x-ray irradiation have all been used with success. It seems worthwhile to record the results obtained so that at a later date it may be possible to evaluate better the various methods now employed in the treatment of this rare disease."

The article is illustrated. W. ZENTMAYER.

CONCERNING DETACHMENT OF THE RETINA. H. S. GRADLE, Am. J. Ophth. **26:** 524 (May) 1943.

Gradle records an interesting case of detachment of the retina occurring in a woman aged 43. Preoperative immobilization of the patient for five to ten days is advised. The position of the patient must be maintained until the surgical procedure is completed. During the latter half of the operation the cornea frequently becomes hazy, so that the necessary view of the fundus is obscured. This was found to be due to pronounced increase in the tension of the eye. The haze disappeared within a few minutes after the final perforating puncture had been made. The hypertension is due to the shrinkage of the sclera by diathermy cauterization, with resultant decrease in the total volume capacity of the eyeball. W. ZENTMAYER.

Tumors

GLIOMA OF THE RETINA IN SUCCESSIVE GENERATIONS. W. L. BENEDICT and E. M. PARKHILL, Am. J. Ophth. **26:** 511 (May) 1943.

In 2 of the 4 cases of glioma of the retina reported the tumor occurred in successive generations. The mother of the patient in case 1 had had a glioma of the right eye at the age of 7 months. The diagnosis of the daughter's lesion was confirmed by microscopic examination. The patients in cases 2 and 3 were twin sisters, and the patient in case 3 subsequently gave birth to a daughter (case 4) who had bilateral glioma of the retina at the age of 2 years and 4 months.

In all 4 cases the diagnosis was confirmed by microscopic examination. The disease was bilateral in cases 1, 3 and 4. In case 2 there was still no evidence of tumor in the right eye at the time of the patient's death.

W. ZENTMAYER.

GLIOMA OF THE RETINA: A REVIEW OF TWELVE CASES. W. B. E. MCCREA, Brit. J. Ophth. **27:** 259 (June) 1943.

McCrea summarizes his clinicopathologic study of 12 patients with so-called glioma of the retina who were treated at the Royal Victoria Eye and Ear Hospital during the period from 1938 to 1942 inclusive.

Incidence.—This series of 12 patients represents an incidence of 1 in a hospital population of 6,126, or 0.0017 per cent. This value is rather lower than has usually been reported in the literature—0.04 to 0.01 per cent (0.009 per cent in forty years at Innsbruck, as reported by Badtke).

Age.—Two thirds of the patients were 3 years old or under when they came under observation,

the ages of the others being 8 (seen originally at the age of 1 year and 10 months), 3½, 4½ and 67 years respectively. The age of the last patient appears to constitute a record—48 years (Verhoeff) and 66 and 35 years (Gerard and Morel) being the highest previously reported.

Sex.—Eight patients were males and 4 females. The figures in the literature indicate that the sexes are affected in about equal numbers.

Heredity.—For none of the patients was it possible to trace any hereditary factor.

Eyes Involved.—The disease was bilateral in 3 patients and unilateral in 9 patients.

Trauma.—Two patients had a history of trauma.

Mortality and Recovery.—Of the 12 patients, 5 were known to have died; 1 could not be traced and was presumed to be dead, and 6 were alive and well at the time of the report, the periods varying from four years to four months since excision of the eye. The time elapsing between the diagnosis and removal of the eye and the patient's death was seven months for patient 1, nine months for patient 2, ten months for patient 6, nine years for patient 7 and five months for patient 8.

Condition of the Optic Nerve and Extraocular Extension.—In only 1 globe (patient 8) was extraocular extension demonstrated. In patients 1 and 2 invasion of the orbital tissue was proved microscopically. The fifth patient was of particular interest in that there was invasion of the optic nerve, as far as the lamina cribrosa, with recovery, the patient being in good health three and a half years later (three years being generally regarded as the length of time necessary before a cure can be regarded as having taken place).

All the patients with involvement of the optic nerve outside the lamina cribrosa died.

Type of Tumor.—Eleven patients had retinoblastoma, 5 of whom died, and 1 had a neuroepithelioma and was alive six months after operation. Parkell and Benedict, in a review of 16 cases each of retinoblastoma and neuroepithelioma in which they had been able to trace the patient, found good results in 12.6 and 62.5 per cent respectively. Badtke, in a review of the cases of glioma at Innsbruck from 1900 to 1939, expressed the opinion that the chance of cure was from 50 to 60 per cent. He placed the rate of recovery in the cases of bilateral glioma at 33 per cent. All the patients with bilateral glioma at the Royal Victoria Eye and Ear Hospital died. W. ZENTMAYER.

ANGIOMA OF THE RETINA. A. G. CROSS, Brit. J. Ophth. **27:** 372 (Aug.) 1943.

The case reported here is of interest as demonstrating a retinal angioma without the excessive gliosis which is characteristic of the condition described by von Hippel. In a woman aged 29 the left eye presented a gray swelling adjoining the nasal margin of the disk. Several dilated veins were present over this tumor. Above and to the nasal side of the swelling there were about half a dozen red spots, which appeared to be retinal hemorrhages. As the lesion increased in size, the original diagnosis of congenital malformation was changed to that of probable malignant growth, and the eye was excised. Pathologic examination revealed a vascular formation at the junction of the retina and the optic disk, invading the optic nerve but not the choroid. There were endothelium-lined channels, containing red blood corpuscles and varying in size from capillaries to vessels as large as the main branches of the central artery and vein. The supporting framework contained glia cells.

The article is illustrated. W. ZENTMAYER.

Vision

ORTHOPTICS: EDUCATION IN BINOCULAR SKILL. J. E. LANCASTER, Am. J. Ophth. **26:** 463 (May) 1943.

Lancaster reaches the following conclusions:

"Orthoptics is not exercising the ocular muscles; it is not even primarily a procedure designed to straighten the eyes. It is the teaching of a patient to use his two eyes together for comfortable binocular vision. The emphasis in such training is the teaching of a skill in the use of unskilled neuromuscular coördination. Orthoptics will be of much greater value to physicians and their patients when its function is re-evaluated: not as primarily concerned with anomalies of eye position, but as devoted to helping the patient learn comfortable binocular visual habits." W. ZENTMAYER.

Therapeutics

IONTOTHERAPY (IONIC MEDICATION, IONTOPHORESIS, IONISATION) AS AN AID IN OPHTHALMIC THERAPEUTICS. N. FEMING, Brit. J. Ophth. **27:** 354 (Aug.) 1943.

Fleming describes the technic and the various agents employed by him in this form of therapy. He summarizes his results as follows:

"The principal feature of treatment by iontotherapy is reduction of congestion and so of inflammation generally.

"It is therefore of use in all forms of conjunctivitis.

"Cases are described showing remarkable success even in the treatment of such deep inflammations as scleritis, irido-cyclitis and retro-bulbar neuritis.

"These claims are based on experience gained in the treatment of some thousand cases, during the last ten years, both in hospital and private practice.

"Iontotherapy is likely to be of particular value in the treatment of war casualties."

W. ZENTMAYER.

Book Reviews

Virus Diseases in Man, Animal and Plant. By Gustav Seiffert. English translation by Marion Lee Taylor, Ph.D. Price, $5. Pp. 323, with 7 illustrations. New York: Philosophical Library, Inc., 1944.

The author states in his preface that it is his purpose to furnish an introduction to the field of virus diseases by reviewing the most recent literature and by summarizing the present status of virus investigation. He points out, quite rightly, that recent surveys of the whole field, or of its parts, have been full of gaps. It is unfortunate, therefore, that his book, while offering a valuable survey of certain aspects of the subject, is itself full of gaps and in general disappointing. The translator is evidently unfamiliar with virus terminology, and what appears to have been an attempt at literal translation has led to some all but unintelligible passages. There are also innumerable errors in spelling and punctuation, evidence of a regrettable lack of care in proofreading.

Workers in this field will find valuable the first section, of ninety pages, which presents a general consideration of virus diseases. The second section consists of reviews of the individual virus diseases, and many of these seem unnecessarily short and incomplete. This is particularly true with respect to the virus diseases of ophthalmologic importance, herpes simplex being the only one receiving any sort of adequate treatment. Perhaps the diseases most poorly treated are trachoma and inclusion blennorrhea, the latter being called, by some error of the translator or the author, "trachoma of the conjunctivae." While the author claims to cover the major research of the last decade, no reference is made to the numerous and valuable studies on trachoma by Julianelle and his associates.

The third part of the volume is concerned with virus-like agents, including the rickettsias and the organisms of pleuropneumonia. The fourth part deals with filtrable forms of bacteria. The concluding section summarizes the methods of virus investigation and is the part which, perhaps, will be most valuable to the student of viruses.

It is unfortunate that a work so needed, and potentially so valuable, should be marred by such poor composition and by so many omissions.

PHILLIPS THYGESON.

Transactions of the American Ophthalmological Society. Volume 41. Pp. 628. Philadelphia: American Ophthalmological Society, 1943.

This volume brings the activities of the society at the annual meeting at Hot Springs, Va., June 10, 11 and 12, 1943, with Dr. H. H. McGuire, president, in the chair. Twenty-two papers were read and discussed. The subjects ranged from the intracapsular cataract operation, keratoconjunctivitis sicca, distribution of certain oxidative enzymes in the ciliary body, split macula, retinal detachment and trauma, hypertensive diseases, pigment freckles of the iris and traumatic enophthalmos to choked disk and low intrathecal pressure. There are, in addition, six obituaries and the theses of nine successful candidates.

The editor, Dr. Wilfred E. Fry, of Philadelphia, deserves great praise for the excellent appearance of this volume.

ARNOLD KNAPP.

News and Notes

GENERAL NEWS

American Board of Ophthalmology.—Examinations during 1945 have been tentatively scheduled for the following months in the cities mentioned, the program being contingent on war and transportation conditions.

Los Angeles in January during the midwinter course, provided the number of applicants warrant it. Applications should be in hand by October 1.

New York in June. The exact dates will be announced about January 1. Applications should be sent in by December 1.

Chicago in October. The exact dates will be announced later. Applications should be in hand by April 1.

The executive office of the board has been changed to Cape Cottage, Maine, and all correspondence should be sent to that address.

The third edition of the "Directory of Medical Specialists," listing the names of and biographic data on all persons certified by the fifteen American boards is to be published early in 1945. Collection of biographic data for the diplomates certified since the 1942 edition and revision of the older listings in that volume are now going forward rapidly. Diplomates are requested to make prompt return of the data regarding their biographies as soon as possible after receiving the proper forms from the publication office soon to be mailed to them.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 17 Horseferry Rd., London, England.

Pan-American Congress of Ophthalmology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretaries: Dr. Conrad Berens, 35 E. 70th St., New York. Dr. M. E. Alvaro, 1511 Rua Consolacão, São Paulo, Brazil.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. W. Clark Souter, 9 Albyn Pl., Aberdeen, Scotland.
Secretary: Dr. Frederick Ridley, 12 Wimpole St., London, W. 1.

Chengtu Ophthalmological Society

President: Dr. Eugene Chan.
Secretary: Dr. K. S. Sun.
Place: Eye, Ear, Nose and Throat Hospital, Chengtu, China.

Chinese Ophthalmology Society

Presdient: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.

Hungarian Ophthalmological Society

President: Prof. I. Imre, Budapest.
Assistant Secretary: Dr. Stephen de Grósz, University Eye Hospital, Máriautca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Place: Birmingham and Midland Eye Hospital.

North of England Ophthalmological Society

President: Dr. A. MacRae, 6 Jesmond Rd., Newcastle-upon-Tyne, England.
Secretary: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Place: Manchester, Bradford, Leeds, Newcastle-upon-Tyne, Liverpool and Sheffield, in rotation. Time: October to April.

Ophthalmological Society of Australia

President: Dr. A. James Flynn, 135 Macquarie St., Sydney.
Secretary: Dr. D. Williams, 193 Macquarie St., Sydney.

Ophthalmological Society of Egypt

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the secretary, Dr. Mohammed Khalil.

Ophthalmological Society of the United Kingdom

President: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Secretary: Mr. L. H. Savin, 7 Queen St., London, W. 1, England.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Rd., Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Oxford Ophthalmological Congress

Master: Mr. P. G. Doyne, 60 Queen Anne St., London, W. 1, England.
Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Place: Oxford, England. Time: July 8-9, 1943.

Palestine Ophthalmological Society

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

Polish Ophthalmological Society

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4. Warsaw.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Col. F. A. Juler, 96 Harley St., London, W. 1, England.
Secretary: Dr. Harold Ridley, 60 Queen Anne St., London, W. 1, England.

SÃO PAULO SOCIETY OF OPHTHALMOLOGY

President: Dr. W. Belfort Mattos, Caixa Postal, 4086, São Paulo, Brazil.
Secretary: Dr. Silvio de Almeida Toledo, Enfermaria Santa Luzia, Santa Casa de Misericordia, Cesario Motta, St. 112, São Paulo, Brazil.

SOCIEDAD ARGENTINA DE OFTALMOLOGIA

Chairman: Dr. Jorge Malbrán, Buenos Aires.
Secretary: Dr. Benito Just Tiscornia, Santa Fe 1171, Buenos Aires.

SOCIEDAD OFTALMOLOGIA DEL LITORAL, ROSARIO (ARGENTINA)

President: Prof. Dr. Carlos Weskamp, Laprida 1159, Rosario.
Secretary: Dr. Juan M. Vila Ortiz, Córdoba 1433, Rosario.
Place: Rosario. Time: Last Saturday of every month, April to November, inclusive. All correspondence should be addressed to the President.

SOCIEDADE DE OPHTHALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DA BAHIA

President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil.
Secretary: Dr. Adroaldo de Alencar, Brazil.
All correspondence should be addressed to the President.

SOCIETÀ OFTALMOLOGICA ITALIANA

President: Prof. Dott. Giuseppe Ovio, Ophthalmological Clinic, University of Rome, Rome.
Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo, 1, Rome.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö.

TEL AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Secretary: Dr. R. J. Masters, 23 E. Ohio St., Indianapolis.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Lawrence T. Post, Metropolitan Bldg., St. Louis.
President-Elect: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Executive Secretary-Treasurer: Dr. William L. Benedict, 101–1st Ave. Bldg., Rochester, Minn.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. S. Judd Beach, 704 Congress St., Portland, Maine.
Secretary-Treasurer: Dr. Walter S. Atkinson, 129 Clinton St., Watertown, N. Y.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

Chairman: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Secretary-Treasurer: Major Brittain F. Payne, School of Aviation Medicine, Randolph Field, Texas.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. Alexander E. MacDonald, 170 St. George St., Toronto.
Secretary-Treasurer: Dr. L. J. Sebert, 170 St. George St., Toronto.

CANADIAN OPHTHALMOLOGICAL SOCIETY

President: Dr. R. Evatt Mathers, 34½ Morris St., Halifax, N. S.
Secretary-Treasurer: Dr. Kenneth B. Johnston, Suite 1, 1509 Sherbrooke St. W., Montreal.
Place: Halifax, N. S. Time: Aug. 4-5, 1944.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. Mason H. Bigelow, 1790 Broadway, New York.
Secretary: Miss Regina E. Schneider, 1790 Broadway, New York.
Executive Director: Mrs. Eleanor Brown Merrill, 1790 Broadway, New York.

SECTIONAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. N. Zwaifler, 46 Wilbur Ave., Newark.
Secretary: Dr. William F. Keim Jr., 25 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. J. Friend, 425 E. Grand Ave., Beloit, Wis.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Paul A. Chandler, 5 Bay State Rd., Boston.
Secretary-Treasurer: Dr. Merrill J. King, 264 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. D. H. O'Rourke, 1612 Tremont Pl., Denver.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. L. L. Bull, 1215–14th Ave., Seattle, Wash.
Secretary-Treasurer: Dr. Barton E. Peden, 301 Stimson Bldg., Seattle 1.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. Sheldon Clark, 27 E. Stephenson St., Freeport, Ill.
Secretary-Treasurer: Dr. Harry R. Warner, 321 W. State St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. M. H. Pike, Midland, Mich.
Secretary-Treasurer: Dr. R. H. Criswell, 407 Phoenix Bldg., Bay City, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. J. C. Decker, 515 Francis Bldg., Sioux City, Iowa.
Secretary-Treasurer: Dr. J. E. Dvorak, 408 Davidson Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. John H. Burleson, 414 Navarro St., San Antonio, Texas.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville, S. C.

SOUTHWESTERN ACADEMY OF EYE, EAR, NOSE AND THROAT

President: Dr. H. L. Brehmer, 221 W. Central Ave., Albuquerque, N. Mex.
Secretary: Dr. A. E. Cruthirds, 1011 Professional Bldg., Phoenix, Ariz.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. W. M. Dodge, 716 First National Bank Bldg., Battle Creek.
Secretary-Treasurer: Dr. Kenneth Lowe, 25 W. Michigan Ave., Battle Creek.
Time: Last Thursday of September, October, November, March, April and May.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Ray Parker, 218 Franklin St., Johnston, Pa.
Secretary-Treasurer: Dr. J. McClure Tyson, Deposit Nat'l Bank Bldg., DuBois.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. Raymond C. Cook, 701 Main St., Little Rock.
Secretary: Dr. K. W. Cosgrove, Urquhart Bldg., Little Rock.

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. C. A. Ringle, 912–9th Ave., Greeley.
Secretary: Dr. W. A. Ohmart, 1102 Republic Bldg., Denver.
Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. F. L. Phillips, 405 Temple St., New Haven.
Secretary-Treasurer: Dr. W. H. Turnley, 1 Atlantic St., Stamford, Conn.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: William O. Martin Jr., Doctors Bldg., Atlanta.
Secretary-Treasurer: Dr. C. K. McLaughlin, 526 Walton St., Macon.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. F. McK. Ruby, Union City.
Secretary: Dr. Edwin W. Dyar Jr., 23 E. Ohio St., Indianapolis.
Place: French Lick. Time: First Wednesday in April.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. K. Von Lackum, 117–3d St. S. E., Cedar Rapids.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Val H. Fuchs, 200 Carondelet St., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Robert H. Fraser, 25 W. Michigan Ave., Battle Creek.
Secretary: Dr. R. G. Laird, 114 Fulton St., Grand Rapids.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Secretary: Dr. William A. Kennedy, 372 St. Peter St., St. Paul.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. William Morrison, 208 N. Broadway, Billings, Mont.
Secretary: Dr. Fritz D. Hurd, 309 Medical Arts Bldg., Great Falls.

Nebraska Academy of Ophthalmology and Otolaryngology

President: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. John Peterson, 1307 N St., Lincoln.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr B. E. Failing, 31 Lincoln Park, Newark.
Secretary: Dr. George Meyer, 410 Haddon Ave., Camden.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Harold J. Joy, 504 State Tower Bldg., Syracuse 2.
Secretary: Dr. Maxwell D. Ryan, 660 Madison Ave., New York 21.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. Hugh C. Wolfe, 102 N. Elm St., Greensboro.
Secretary: Dr. Vanderbilt F. Couch, 104 W. 4th St., Winston-Salem.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. L. Diven, City National Bank Bldg., Bismarck.
Secretary-Treasurer: Dr. A. E. Spear, 20 W. Villard, Dickenson.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. Paul Neely, 1020 S. W. Taylor St., Portland.
Secretary-Treasurer: Dr. Lewis Jordon, 1020 S. W. Taylor St., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

Pennsylvania Academy of Ophthalmology and Otolaryngology

President: Dr. Lewis T. Buckman, 83 S. Franklin St., Wilkes-Barre.
Secretary Pro Tem: Dr. Paul C. Craig, 232 N. 5th St., Reading.
Time: Last week in April.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. J. L. Sanders, 222 N. Main St., Greenville.
Secretary: Dr. J. H. Stokes, 125 W. Cheves St., Florence.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. Wesley Wilkerson, 700 Church St., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. F. H. Rosebrough, 603 Navarro St., San Antonio.
Secretary: Dr. M. K. McCullough, 1717 Pacific Ave., Dallas.

Utah Ophthalmological Society

President: Dr. R. B. Maw, 699 E. South Temple, Salt Lake City.
Secretary-Treasurer: Dr. Charles Ruggeri Jr., 1120 Boston Bldg., Salt Lake City.
Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Mortimer H. Williams, 30½ Franklin Rd. S. W., Roanoke.
Secretary-Treasurer: Dr. Meade Edmunds, 34 Franklin St., Petersburg.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. E. L. Mather, 39 S. Main St., Akron, Ohio.
Secretary-Treasurer: Dr. V. C. Malloy, 2d National Bank Bldg., Akron, Ohio.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. B. M. Cline, 153 Peachtree St. N. E., Atlanta, Ga.
Acting Secretary: Dr. A. V. Hallum, 478 Peachtree St. N. E., Atlanta, Ga.
Place: Grady Hospital. Time: 6:00 p. m., fourth Monday of each month, from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Ernst Bodenheimer, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Thomas R. O'Rourk, 104 W. Madison St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to March.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. Luther E. Wilson, 919 Woodward Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. William B. Agan, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Benjamin C. Rosenthal, 140 New York Ave., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Walter F. King, 519 Delaware Ave., Buffalo.
Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. Douglas Chamberlain, Chattanooga Bank Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Vernon M. Leech, 55 E. Washington St., Chicago.
Secretary: Dr. W. A. Mann, 30 N. Michigan Ave., Chicago.
Place: Chicago Towers Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati General Hospital Ophthalmology Staff

Chairman: Dr. D. T. Vail, 441 Vine St., Cincinnati.
Secretary: Dr. A. A. Levin, 441 Vine St., Cincinnati.
Place: Cincinnati General Hospital. Time: 7:45 p. m., third Friday of each month except June, July and August.

Cleveland Ophthalmological Club

Chairman: Dr. Shandor Monson, 1621 Euclid Ave., Cleveland.
Secretary: Dr. Carl Ellenberger, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Clerk: Dr. George F. J. Kelly, 37 S. 20th St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. H. D. Emswiler, 370 E. Town St., Columbus, Ohio.
Secretary-Treasurer: Dr. D. G. Sanor, 206 E. State St., Columbus, Ohio.
Place: The Neil House. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. Arthur Padillo, 414 Medical Professional Bldg., Corpus Christi, Texas.
Secretary: Dr. Edgar G. Mathis, 815 Medical Arts Bldg., Corpus Christi, Texas.
Time: Second Friday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Ruby K. Daniel, Medical Arts Bldg., Dallas 1, Texas.
Secretary: Dr. Tom Barr, Medical Arts Bldg., Dallas 1, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. H. C. Schmitz, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Byron M. Merkel, 604 Locust St., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Dr. Raymond S. Goux, 545 David Whitney Bldg., Detroit 26.
Secretary: Dr. Arthur Hale, 1609 Eaton Tower, Detroit.
Place: Club rooms of Wayne County Medical Society. Time: 6:30 p. m., third Thursday of each month, November through April.

Detroit Ophthalmological Society

President: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. Leland F. Carter, 1553 Woodward Ave., Detroit.
Place: Club rooms of Wayne County Medical Society. Time: Third Thursday of each month from November to April, inclusive.

Eastern New York Eye, Ear, Nose and Throat Association

President: Appointed at each meeting.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Rex Howard, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Gough, Medical Arts Bldg., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Ophthalmological and Oto-Laryngological Section

President: Dr. Lyle J. Logue, 1304 Walker Ave., Houston, Texas.

Secretary: Dr. John T. Stough, 803 Medical Arts Bldg., Houston, Texas.

Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. Myron Harding, 23 E. Ohio St., Indianapolis.

Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.

Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Edgar Johnson, 906 Grand Ave., Kansas City, Mo.

Secretary: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.

Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Harold Snow, 614 S. Pacific Ave., San Pedro, Calif.

Secretary-Treasurer: Dr. Oliver R. Nees, 508 Times Bldg., Long Beach, Calif.

Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. M. E. Trainor, 523 W. 6th St., Los Angeles.

Secretary-Treasurer: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.

Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye and Ear Society

President: Dr. Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.

Secretary-Treasurer: Dr. J. W. Fish, 321 W. Broadway, Louisville, Ky.

Place: Brown Hotel. Time. 6:30 p. m., second Thursday of each month from September to May, inclusive.

Lower Anthracite Eye, Ear, Nose and Throat Society

Chairman: Each member in alphabetical order.

Secretary: Dr. James J. Monohan, 31 S. Jardin St., Shenandoah, Pa.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. P. S. Constantinople, 1835 I St. N. W., Washington.

Secretary: Dr. Frazier Williams, 1801 I St. N. W., Washington.

Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member in alphabetical order.

Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.

Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month from September to May.

Milwaukee Oto-Ophthalmic Society

President: Dr. Edwin C. Bach, 324 E. Wisconsin Ave., Milwaukee.

Secretary-Treasurer: Dr. Ralph T. Rank, 238 W. Wisconsin Ave., Milwaukee.

Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Montgomery County Medical Society

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.

Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.

Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. J. Rosenbaum, 1396 Ste. Catherine St. W., Montreal, Canada.

Secretary: Dr. L. Tessier, 1230 St. Joseph Blvd. E., Montreal, Canada.

Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. M. M. Cullom, 700 Church St., Nashville, Tenn.

Secretary: Dr. R. E. Sullivan, 432 Doctors Bldg., Nashvile, Tenn.

Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May

New Haven Ophthalmological Society

President: Dr. William H. Ryder, 185 Church St., New Haven, Conn.

Secretary: Dr. Frederick A. Wiess, 255 Bradley St., New Haven, Conn.

New Orleans Ophthalmological and Oto-Laryngological Society

President: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.

Secretary: Dr. Mercer G. Lynch, 1018 Maison Blanche Bldg., New Orleans.

Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to May.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Frank C. Keil, 660 Madison Ave., New York.

Secretary: Dr. Willis S. Knighton, 121 E. 61st St., New York.

Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Milton Berliner, 57 W. 57th St., New York.

Secretary: Dr. Benjamin Esterman, 983 Park Ave., New York.

Place: Squibb Hall, 745–5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Oklahoma City Academy of Ophthalmology and Otolaryngology

President: Dr. James P. Luton, 117 N. Broadway, Oklahoma City.

Secretary: Dr. Harvey O. Randel, 117 N. Broadway, Oklahoma City.

Place: University Hospital. Time: Second Tuesday of each month from September to May.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. D. D. Stonecypher, Nebraska City, Neb.

Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.

Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m. dinner; 7 p. m. program; third Wednesday of each month from October to May.

Passaic-Bergen Ophthalmological Club

President: Dr. Thomas Sanfacon, 340 Park Ave., Paterson, N. J.

Secretary-Treasurer: Dr. J. Averbach, 435 Clinton Ave., Clinton, N. J.

Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

President: Dr. Isaac Tassman, 136 S. 16th St., Philadelphia.

Secretary: Dr. Glen Gregory Gibson, 255 S. 17th St., Philadelphia.

Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. John B. McMurray, 6 S. Main St., Washington, Pa.

Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.

Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Reading Eye, Ear, Nose and Throat Society

President: Dr. R. M. Brickbauer, Shillington, Pa.

Secretary: Dr. Paul C. Craig, 232 N. 5th St., Reading, Pa.

Place: Wyomissing Club. Time: 6:30 p. m., third Wednesday of each month from October to July.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. Peter N. Pastore, Medical College of Virginia, Richmond, Va.

Secretary: Dr. Clifford A. Folkes, Professional Bldg., Richmond, Va.

Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. Frank Barber, 75 S. Fitzhugh St., Rochester, N. Y.

Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.

St. Louis Ophthalmic Society

President: Dr. C. C. Beisbarth, 3720 Washington Blvd., St. Louis.

Secretary: Dr. H. R. Hildreth, 508 N. Grand Blvd., St. Louis.

Place: Oscar Johnson Institute. Time: Clinical meeting, 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmo-Oto-Laryngological Society

President: Dr. Belvin Pritchett, 705 E. Houston St., San Antonio 5, Texas.

Secretary-Treasurer: Lt. Col. John L. Matthews, AAF School of Aviation Medicine, Randolph Field, Texas.

Place: San Antonio, Brooke General Hospital, Randolph Field or San Antonio Aviation Cadet Center. Time: 7 p. m., second Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Roy H. Parkinson, 870 Market St., San Francisco.

Secretary: Dr. A. G. Rawlins, 384 Post St., San Francisco.

Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.

Secretary-Treasurer: Dr. Kenneth Jones, Medical Arts Bldg., Shreveport, La.

Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Clarence A. Veasey Sr., 421 W. Riverside Ave., Spokane, Wash.
Secretary: Dr. Clarence A. Veasey Jr., 421 W. Riverside Ave., Spokane, Wash.
Place: Spokane Medical Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. A. H. Rubenstein, 713 E. Genesee St., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. H. Blaisdell, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toledo Eye, Ear, Nose and Throat Society

Chairman: Dr. E. W. Campbell, 316 Michigan St., Toledo, Ohio.
Secretary: Dr. L. C. Ravin, 316 Michigan St., Toledo, Ohio.
Place: Toledo Club. Time: Each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. W. R. F. Luke, 316 Medical Arts Bldg., Toronto, Canada.
Secretary: Dr. W. T. Gratton, 216 Medical Arts Bldg., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. S. Bockoven, 1752 Massachusetts Ave., Washington, D. C.
Secretary-Treasurer: Dr. John Lloyd, 1218–16th St. N. W., Washington, D. C.
Place: Medical Society of District of Columbia Bldg., 1718 M St. N. W., Washington, D. C. Time: 7:30 p. m., first Monday in November, January, March and May.

Wilkes-Barre Ophthalmological Society

Chairman: Each member in turn.
Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.
Place: Office of chairman. Time: Last Tuesday of each month from October to May.